THE EYE

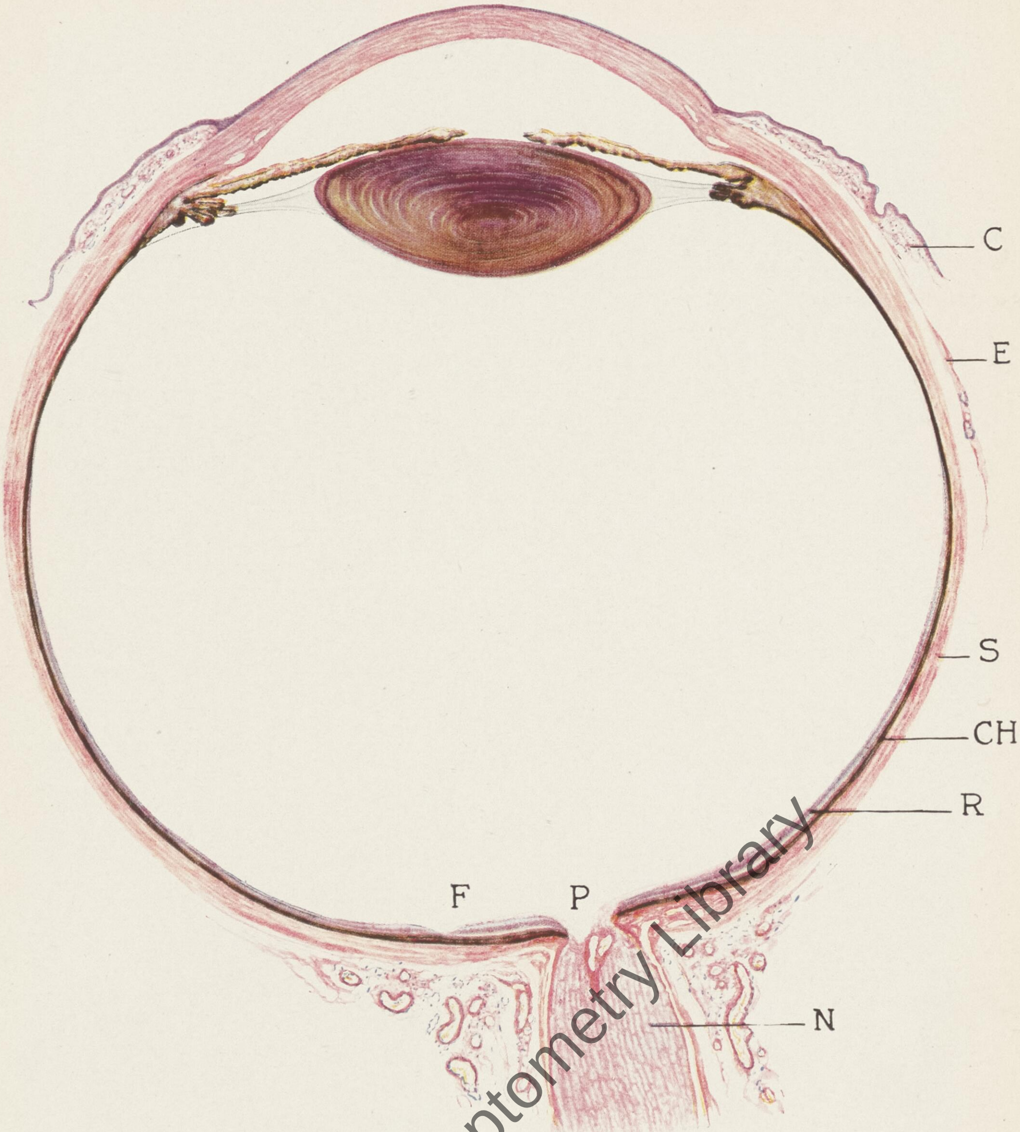

PLATE I.—THE STRUCTURES OF THE GLOBE IN HORIZONTAL CROSS SECTION, LEFT EYE.

C, conjunctiva; *E*, episclera; *S*, sclera; *CH*, choroid; *R*, retina; *N*, optic nerve; *F*, fovea; *P*, porus opticus. The ciliary body and processes, iris, crystalline lens, zonular fibers, canal of Schlemm and cornea are not lettered.

THE EYE

BY

C. W. RUTHERFORD, M.D., F.A.C.S.

ASSOCIATE IN OPHTHALMOLOGY, INDIANA UNIVERSITY SCHOOL OF MEDICINE; FELLOW OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY; MEMBER OF STAFFS OF THE UNIVERSITY HOSPITALS, THE INDIANAPOLIS CITY HOSPITAL, AND THE INDIANAPOLIS CITY DISPENSARY.

WITH 305 BLACK AND WHITE FIGURES AND
12 ORIGINAL COLORED PLATES

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PREFACE

The needs of the general practitioner and student of medicine have had first consideration in the preparation of this text. The arrangement of the contents facilitates systematic study, and each subject is complete under the plan of presentation. The advantages of a work of reference and of a teaching text are combined.

The practice of ophthalmology is based upon the broad foundations of clinical medicine. The visual apparatus is a part of the body, and not a dissociated feature. The structures of the eye and the orbit may express their own diseases primarily, they may be affected secondarily, and they may, by peculiar behavior, guide the diagnostician to a remote lesion.

Diseases of the eye obey common laws. Their successful management requires some knowledge of the eye and its environment. For that reason, the essentials of the anatomy have been stated as an introduction to each section. The commoner diseases of each structure have been described carefully, while those of less frequent occurrence have been discussed as an aid to differential diagnosis. Free references have been made to ocular evidences of general diseases, since they are important in diagnosis. The latest established views and opinions have been presented, while controversial discussions occupy little space.

The text is the product of the experience of the author and of the teaching of those who have contributed to the advancement of medical knowledge by personal communications, in literature, and at the sessions of the learned societies. Few direct references have been made.

The author sincerely appreciates the opportunities that have been accorded him by the Department of Ophthalmology and by other departments during the period of his connection with Indiana University School of Medicine.

C. W. Rutherford.

Indianapolis

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CHAPTER I

THE EYELIDS

ESSENTIALS OF THE ANATOMY

The function of the eyelids is to protect the structures behind them, to cleanse and moisten the cornea, and to exclude light from the retina. The lashes and sensory nerves of the lid margins give added protection. The lids are designated as superior or upper, and inferior or lower. The superior is broader vertically and covers the larger surface area of the eye; it has an elevator muscle which gives it greater motility than is possessed by the inferior.

Each lid has an attached and a free border, a lateral (outer, external or temporal) and a medial (inner, internal or nasal) extremity, and an anterior or cutaneous, and a posterior or conjunctival surface. Anatomically, the lids are palpebræ. The opening between the lids is the palpebral aperture or fissure; the lateral and the medial extremity of the aperture is a canthus or angle. The tissues of the two lids are united at either extremity by a commissure.

Embryologically, the lids are formed by a folding of skin which encloses muscular, vascular, and fibrous tissues in the developmental processes. The outer layer retains its character as skin, while the inner becomes mucous membrane or conjunctiva. In the formation of each lid a part of the fibrous tissue is modified and compacted into a curved plate or tarsus.

Surrounding the eye is an oval area limited above by the eyebrow and below by the heads of origin of the quadratus labia superioris muscle (Fig. 1). This area becomes apparent in age, in emotional expression, and when the lower lid sags from fatigue or from paralysis. The medial border of the area indicates the location of the lacrimal sac and the opening of the nasolacrimal duct. Outside this area the superficial fascia is dense and contains fat. Within it the skin possesses peculiar characteristics. It is thinner, is less firmly attached by its loose areolar tissue to the muscle beneath, its corium contains no fat, its hairs are very fine and short, and its sweat and sebaceous glands are

small and supply little secretion. In plastic surgery these characteristics are important.

Several skin folds of interest are found in the lids. Each lid has an orbital and a palpebral portion. The palpebral portion coincides with the conjunctival sac, while the orbital part extends from this boundary to beyond the margins of the orbit. At the junction of these portions there is a folding in of the skin (Fig. 1). (See Plate II.) The infolds correspond to the superior border of the upper and the inferior border of the lower tarsal plates. These are the supratarsal and infratarsal folds. They are landmarks for performing eversions of the lids when examining the palpebral conjunctiva,

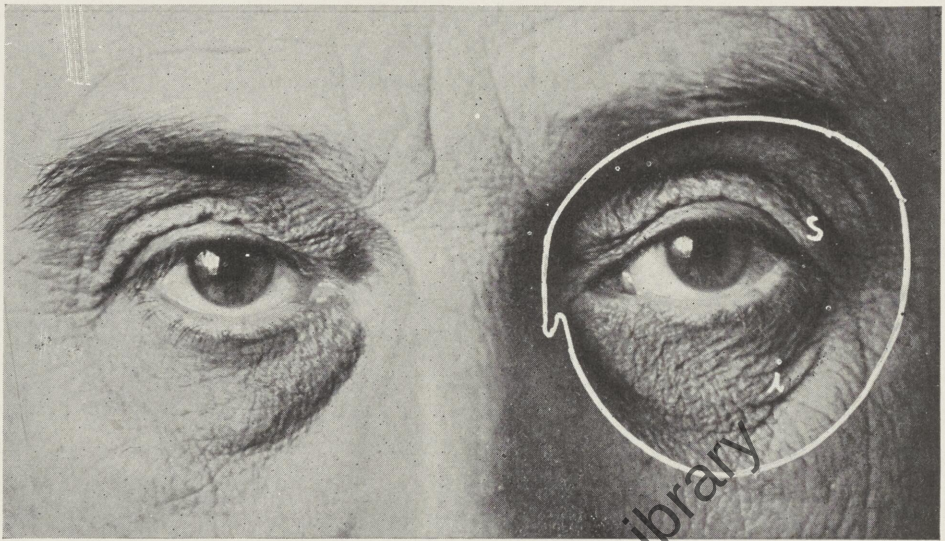


Fig. 1.—Cutaneous Area of the Eyelids.
s, supratarsal fold; *i*, infratarsal fold.

and for some surgical procedures such as the correction of ptosis. During sleep and where the eyes are prominent or the lids swollen, these folds may be obliterated. When the lids are widely opened and while looking upward, the supratarsal folds are accentuated.

In age, when the skin of the lid has lost elasticity, the orbital portion overhangs the palpebral and forms an outfold or pouch. When the redundancy is conspicuous it is called "ptosis adiposa"; it disappears when the eyebrow is drawn upward, thus differentiating it from ptosis due to an accumulation of fluids or fat, or from hypertrophy of the lid. The infold of the lower lid is usually absent in the adult, although present in the infant.

The palpebral aperture may be shortened horizontally. Epicanthus is a fold of skin extending from the bridge of the nose over the inner canthi of

both eyes. It is common to the Mongolian race. A similar fold at the outer canthus of either or both eyes may exist; sometimes this also is called epicanthus. The angle of the outer canthus is narrower than that of the inner, which is widened by the caruncle and the lacus lacrimalis.

The vertical width of the palpebral aperture depends on the anteroposterior position of the globe, and the tone and activity of the muscles of the lids. The aperture is widened when the lids are retracted in exophthalmos, or after the instillation of cocain solutions, and in proptosis.

The aperture is narrowed after enucleation of the globe, often so even with the wearing of a glass eye; when the globe recedes into the orbit (enophthalmos); in ptosis of the upper lid; where the upper lid is hypertrophied, extravasated with blood, infiltrated with serum or inflated with air; as a consequence of some of the complications of trachoma; voluntarily as a protection from wind, dust and bright light; reflexly from the presence of a foreign body on or ulcer of the cornea or conjunctiva; and in blepharospasm.

The skin of the lids is elastic and freely movable, because it is sparingly attached to the underlying muscles by a layer of fine connective tissue fibers which correspond to the denser superficial fascia in other localities. Being surrounded by dense fascial boundaries, the lid area marks the usual limits of a "black eye" from extravasated blood, edema or cellulitis of the lids, and emphysema or air inflation that may follow vigorous blowing of the nose or violent sneezing.

The orbicularis oculi is a voluntary muscle which extends from beyond the anterior surface of the orbital margin to the palpebral aperture. Its function is to close the lids. It consists of three parts: The pars orbitalis, the pars palpebralis, and the pars lacrimalis. The last is described in connection with the lacrimal apparatus.

The pars orbitalis arises from the anterior surface of the orbital rim in the superior portion of its medial third, and from the medial palpebral ligament. Its fibers follow around the orbit and are inserted into the orbital rim in a corresponding situation below. Its function is to support and protect the contents of the orbit, and to reinforce the action of the pars palpebralis. The latter muscle is divided into an upper and a lower portion, corresponding to the respective lids. It is the part necessary to the protection of the globe. It arises from the medial orbital margin and from the medial palpebral ligament; each part traverses its respective lid laterally, and the two parts unite in the lateral palpebral raphé.

The main body of the palpebral muscle lies in two zones; one zone corresponds to the tarsal plates, and the other to the orbital septum. The surface markings of the junction of these zones are the supratarsal and infratarsal folds.

This part of the orbicularis oculi has three functions: voluntary closure of the palpebral aperture, automatic winking, and reflex blinking in bright light.

The muscle of Riolan is a small bundle of fine fibers which is imbedded near the free margin of each lid. It has relations with the ducts of the meibomian glands, and with the hair-follicles and the ducts of their glands.

The levator palpebræ superioris is the remaining voluntary muscle of the lids. It arises from above the optic foramen, and follows the orbital roof to near the midline of the superior margin of the orbit; here it expands as a fan-shaped aponeurosis to be inserted in a variety of ways. Fibers are reflected upward and downward to be inserted into the skin of the upper lid throughout its breadth, necessarily traversing the bundles of the orbicularis oculi to gain their destination. Other fibers take a deeper course and are inserted into the upper border and the anterior surface of the tarsus; these are surgically important. Some fibers mingle with those of the orbital septum, and the most posterior enter the ocular conjunctiva above the cornea.

The marginal fibers are more compact and are inserted at the extreme medial and lateral margins of the orbit. The lateral band divides the lacrimal gland into an orbital and a palpebral portion.

The attachments are mainly for the voluntary elevation of the upper lid. Involuntary fibers, controlled by the sympathetic, are incorporated among those attached to the superior margin of the tarsus. The surface marking of this insertion is the supratarsal fold. These fibers constitute the palpebral muscle of Müller, which maintains elevation of the lid; it is not to be confused with any other muscle of that name. A similar muscle is found in the lower lid.

The orbital septum is a flexible membrane attached to the whole circumference of the orbital rim. It blends with the anterior surface of the inferior tarsus, and with the fibers of the levator palpebræ superioris over the superior tarsus. It is composed of elastic and connective tissue fibers, and forms a diaphragmatic barrier between the orbit and the lids. Blood-vessels and nerves perforate it along its periphery, and by way of these perforations fluids and air can pass from either side to the other when under sufficient pressure.

The tarsi are flexible plates of compact fibrous tissue. The superior gives form to the upper lid, and its posterior concave surface is adapted to the contour of the eyeball. It is much broader than that in the lower lid; the latter is narrow and bandlike. The tarsi are supported medially and laterally by bands which blend at the commissures. The medial extension enters into the medial palpebral ligament, which is of interest in operations on the lacrimal sac; the lateral extension enters into the lateral palpebral raphé, which is divided in performing a canthotomy.

The tarsi support the meibomian glands and the follicles of the cilia (Plate

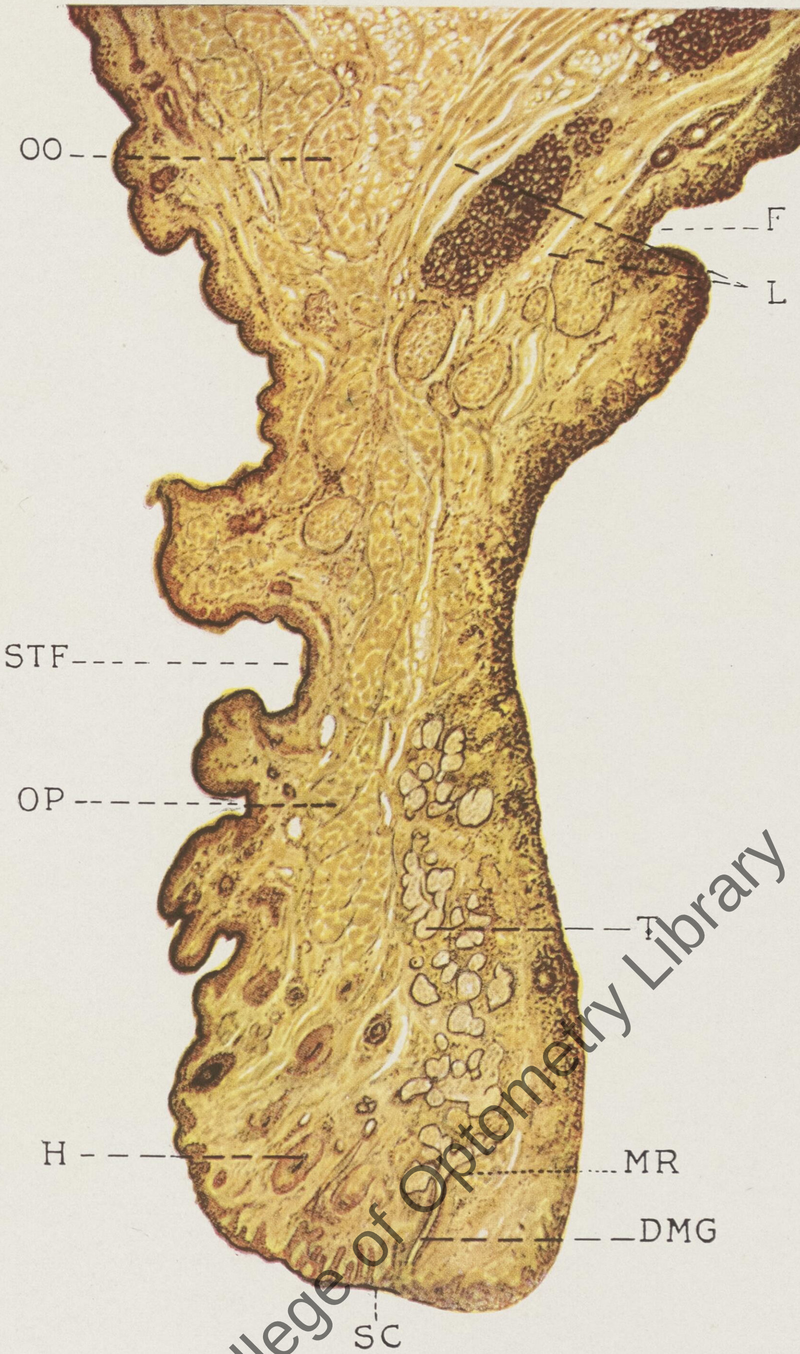


PLATE II.—CROSS SECTION OF CADAVER UPPER EYELID.

OO and *OP*, orbital and palpebral portions of the orbicularis oculi; *F*, fornix; *L*, levator palpebræ superioris; *STF*, supratarsal fold; *T*, tarsus with meibomian glands; *H*, hair-follicle; *MR*, musculus Riolani; *DMG*, duct of meibomian gland; *SC*, transition from conjunctiva to skin.

II). The meibomian glands are large sebaceous glands which are demonstrable in some subjects in the conjunctival surfaces of the lids. There are thirty to forty in the upper lid, and about ten fewer in the lower. They lie parallel in one plane, occupy a direction at right angles to the free margin of the lid, and are limited in extent to the tarsal area (Fig. 2). The orifices of their ducts lie in a single row from the punctum laterally behind the transition from skin to conjunctiva on the free margin of the lid (Fig. 3). The yellowish secretion from these glands is of a fatty nature; it lubricates the margins of the lids so that they do not adhere to each other, and it directs the tears toward the inner canthus for drainage into the nose.

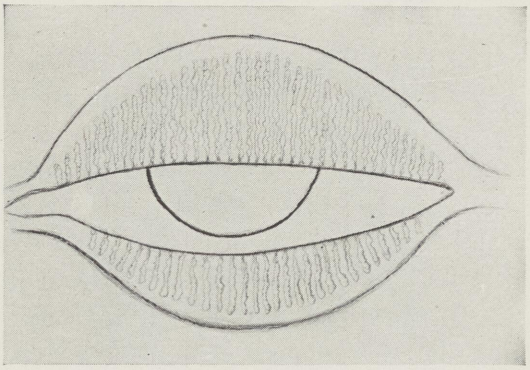


Fig. 2.—Arrangement of Meibomian Glands.

The free margins of the lids are especially constructed. The anterior skin surface is rounded, and bears upon its convexity a row or two of lashes or cilia which are supplied by the sebaceous glands of Zeis and the sweat-glands of Moll. These cilia are short and thick; the upper are curved down and out, and the lower out and down; when the lids are closed the cilia of the two lids are in contact. The follicles of these hairs are well supplied with sensory nerves and blood-vessels.

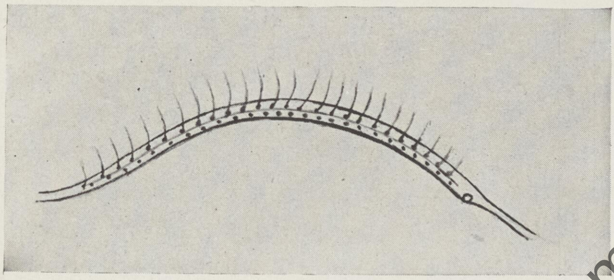


Fig. 3.—Orifices of Meibomian Glands. (After Parsons.)

Immediately in front of the orifices of the meibomian glands is a faint white or gray line. This is to be followed in operations which split the lid into two layers. The skin and muscle are anterior to the line, while the tarsus and conjunctiva are posterior to it. Behind the orifices of the glands the lid margins are flattened for better apposition.

The lacus lacrimalis occupies the medial sixth of the palpebral aperture. In this region the lid margins are free of cilia and their glands. The space is occupied by the caruncle, a small reddish body derived from the lower lid in embryonic life; it directs the tears toward the puncta.

The arterial blood supply is from branches of the external and internal carotids which appear at the circumference of the orbital opening. The branches of the external carotid are the external maxillary (O.T. facial) to the medial side of the orbit, the infra-orbital (continuation of the internal maxillary) below, and several small branches from the superficial temporal to the lateral side. Branches of the ophthalmic division of the internal carotid appear from beneath the superior margin of the orbit.

The external maxillary approaches the eye along the nasojugal fold; at the medial angle of the lids it is the angular artery. This must be considered in operations on the lacrimal sac. The external maxillary anastomoses with the transverse facial and the infra-orbital, and is continuous with the nasal branch of the ophthalmic artery. Branches from the superficial temporal anastomose with branches from other arteries within and without the orbit.

The principal lid branches from the ophthalmic are the nasal, frontal, supra-orbital, lacrimal and palpebral. These also anastomose with other arteries. The integument and muscles of the lids are supplied by all the vessels mentioned. Palpebral branches are derived from the lacrimal and nasal to supply the deeper tissues of the lids by means of vessels near each border of each tarsus. In front of and behind each plate are plexuses derived from these border vessels in common. From the superior of these tarsal arteries, small branches go to the conjunctiva of the fornix and to that of the globe. Small twigs supply the lid margins, which are subject to congestion with marked redness.

The supra-orbital vein lies beneath the superior margin of the orbit and parallel with it. The frontal vein accompanies the supra-orbital artery. The union of these two forms the angular, which in turn becomes the anterior facial vein that accompanies the external maxillary artery. A branch of the supra-orbital passes through the supra-orbital notch or foramen to join the superior ophthalmic; in the notch it receives the frontal diploic vein. The superior ophthalmic also receives a branch from the angular vein.

The anterior facial is in communication with the pterygoid plexus by way of the deep facial vein. The pterygoid plexus is also in communication with the inferior ophthalmic vein, and both the ophthalmics usually enter the cavernous sinus. The veins of the superficial structures communicate with the cavernous sinus by two routes; infections of the sinuses, nose, face or eyelids may be carried to intracranial structures. The superficial and deep veins of the lids anastomose like the arteries. All the venous blood from the regions described empties into the external and internal jugular veins.

The lymph-vessels are superficial and deep. The inferomedial and medial palpebral regions drain toward the submaxillary nodes, while the superolateral

and lateral drain toward the parotid nodes. The latter drains the larger area of the palpebral conjunctiva.

The nerve supply of the eyelids is sensory, motor and sympathetic. The sensory supply is by branches of the ophthalmic division of the trigeminus, and by a few branches from the infra-orbital branch of the maxillary division. The motor supply to the orbicularis oculi is by the facial, and to the levator palpebræ superioris by the motor oculi. The facial is distributed by temporal and zygomatic branches above and below respectively. The sympathetic supplies the plain muscle tissue in each lid.

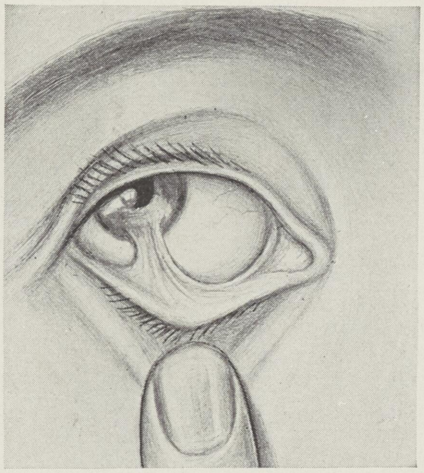


FIG. 4.—SYMBLEPHARON.

Cryptophthalmia is a congenital condition in which the eyelids have not been differentiated; the skin passes from the eyebrow to the cheek without aperture or cilia. A rudimentary eyeball may or may not be present.

Blepharophimosis is a partial closure of the palpebral aperture due to adhesions between the margins of the lids. Ankyloblepharon is a closure of the aperture by adhesions of the lid borders. Symblepharon is an adhesion of the conjunctival surface of the lid to that of the globe (Fig. 4). Ablepharon is a partial or complete absence of the lids, and the globe is exposed. It is usually accompanied by a defective eyeball.

THE EFFECTS OF GENERAL DISEASES ON THE LIDS

The tissues of the eyelids are not essentially different from similar tissues elsewhere, and they are subject to the same diseases. Only because of the proximity of the delicate tissues of the eyeball will treatment require modification, which in a general way it does not.

Erysipelas is a grave disease when it involves the lids, for here the anatomical structure is favorable to vascular congestion and edematous swelling, conditions which are ideal for streptococci. Extension along the sheath of the optic nerve can reach the cranial cavity and terminate the process by a fatal meningitis. By way of the veins an equally fatal cavernous sinus thrombosis can result. Treat the erysipelas, and keep the conjunctival sac clean.

The Exanthemata do not require special treatment unless an ocular structure is involved, such as the occurrence of a corneal ulcer in measles. In these cases the ocular lesion must be managed according to its nature.

Eczema requires energetic treatment if complications are to be avoided; these arise in consequence of scarring. Any disease of the eye or lid accompanied by an overflow of tears to the cheek may cause, aggravate or complicate an eczema, on account of the constant presence of moisture on the skin. Any failure of tear drainage due to altered secretions, false positions of the puncta, or occlusions in the excretory passages must be corrected. The eczema is treated by painting with solution of silver nitrate 5 per cent, or by the application of an ointment of ammoniated mercury 2 per cent, zinc oxid 2 per cent, or ichthyol 5 per cent. These should be applied on gauze.

Fig. 5.—Herpes Zoster Ophthalmicus. (Robert Long Hospital Patient.)

Herpes Zoster Ophthalmicus presents the characteristics of the disease when found elsewhere. Vesicles with inflamed bases appear over the course of the trigeminus nerve and its branches. The eruption follows the branches of the ophthalmic division usually, seldom those of the maxillary, and very rarely those of the mandibular (Fig. 5). The frontal nerve is most frequently involved, although others may be associated with it or affected independently. When vesicles appear on the conjunctiva or cornea the nasociliary branch is involved.

The disease is usually considered to be a neuritis. Central lesions have been found in the gasserian ganglion and its sheath; these consist of inflammations and hemorrhages which are followed by degeneration. The changes are identical with those found in the posterior roots of the cord when the trunk has been the site of the disease. It is practically always limited to one side of the head, which distinguishes it from herpes febrilis and some other

eruptions. The complications consist of corneal lesions, iridocyclitis, glaucoma and panophthalmitis. The duration varies from two weeks to five months.

Treatment is symptomatic. Morphin may be required for pain. The vesicles must not be opened, but protected with dusting powders. Large doses of quinin have been recommended. Some cases respond favorably to light applications of the galvanocautery over the course of the affected nerve. Pitted scars usually follow healing.

Blastomycosis and Sporotrichosis of the lids are rare fungoid diseases that can be identified by the successful culture and recognition of the fungi. The former is a flat, wartlike, crust-covered multiple lesion bounded by an elevated red margin. The latter is a granulomatous lesion accompanied by swelling of neighboring lymph-nodes; it is not to be confused with Parinaud's conjunctivitis. Both have been mistaken for epithelioma, syphilis and tuberculosis of the lids. Radium and x-ray have been used. Potassium iodid to toleration has been most effective.

The use of radium in the vicinity of the eye is not advised, unless it can be employed by an expert. Disastrous consequences follow unskilled employment of this agent.

Syphilis may attack the lids either as a primary lesion, or as one of the later forms of cutaneous manifestations. The primary ulcer has the typical punched-out appearance and indurated raised edges; in this location it may be tender and painful, although it is not ordinarily so. Aside from constitutional treatment, such an ulcer should be kept clean and have antiseptic dressings. A gumma may resemble a chalazion, but it has a tendency to ulcerate like a primary lesion, and to involve deeper tissues.

Tuberculosis and syphilis of the lids are often accompanied by enlarged lymph glands, especially the submaxillary and the preauricular. The former disease is seldom if ever primary to the lids. Scrofula usually overlies an area of carious bone.

Lupus spreads from contiguous surfaces; it may invade the conjunctiva and produce intractable scarring.

Ulcers also result from burns and infected injuries; the usual treatment is required, with due attention to protecting the eyeball by ointments made up with petrolatum as a base.

Xanthelasma is a small oval or round "inlay" which looks like chamois skin, and which usually appears at the inner end of the lid. These are ordinarily symmetrical, and all four lids may be affected. They have cosmetic importance only. They may be excised, but are prone to recur.

Edema of the Lids occurs readily because of the loose attachment of the skin to the muscle beneath. Inflammatory types are distinguished from the

noninflammatory by the usual signs. If possible, the cause of an edema should be determined. A superficial localized mass will move with the skin, while a deeper one will not. The demonstration of a mass differentiates the localized process from one of diffuse character. The position of a localized inflammation may indicate the source of the edema.

A prelacrimal or lacrimal abscess and an acute dacryocystitis will always occur in the region of the tear sac. A *furuncle* occurs on the lid surface, but not at the margin, while a *hordeolum* occurs at the lid margin in connection with the follicle of an eyelash or the duct of a meibomian gland. *Malignant pustule* and *abscess* may occur at any part of the lid. The edema associated with *blepharospasm* may be suspected when the lids can be separated only with great difficulty; this edema resembles that caused by simple venous engorgement.

Extravasations of blood will be recognized by the discoloration of the skin. *Traumatisms* likewise produce discolorations. Edema from erysipelas, eczema and like causes is diffuse; in these cases the skin may be glistening and red. The edema due to heart or kidney diseases is apt to be "puffy," influenced by gravity, and not uniform in intensity; in appearance, this resembles the lower lid edema that follows loss of sleep or exhaustion. *Angioneurotic edema* is intermittent and has the usual characteristics of that symptom when observed elsewhere.

Fig. 6.

If no cause for an edema can be found by external examination, the conjunctiva and eyeball must be investigated. This may be difficult; aside from the clumsy thickening of the lids, muscular spasm or tenderness often complicate the situation. By separating the lids with the fingers or a lid hook (Fig. 6), a few drops of 4 per cent cocain solution can be instilled. The lids may be separated more widely at a second attempt, and a few more drops of the solution can be instilled. If this plan fails, the orbicularis oculi may be paralyzed by injecting novocain solution into the branches of the facial nerve on the temporal side of the orbit; morphin sulphate may be added for pain.

Having separated the lids, a conjunctivitis of the purulent type may be found. The physician should protect himself against the possibility of secretions spurting from the eye of the patient on to his own person, and especially into his own eyes; he should wear glasses and stand to one side of the patient. After novocain has been injected the eye should have an occluding dressing, for when the lids fail to close, the cornea is exposed to injury.

Edema may be caused by panophthalmitis, iridocyclitis, tenonitis, acute

glaucoma, tumor or inflammations of the orbit, or thrombosis of the cavernous sinus; insect bites and stings also cause it. While the edema is advancing or is at its maximum, the skin will be tense and smooth; as soon as it begins to subside, fine horizontal wrinkles appear in the skin. Incomplete edemas at the upper inner angle of the orbit are often due to disease of the frontal sinus; at the lower inner angle, to disease of the maxillary antrum or of the ethmoid cells.

Treatment.—Cold applications are usually more soothing than hot. If the edema is due to emphysema or air inflation, the patient must be warned against blowing his nose, and should be advised to draw nasal secretions into the pharynx.

Molluscum Contagiosum is an affection of the sebaceous glands of the skin. The tumor is discrete, umbilicated, and is not deeply attached. It is said to be contagious. Incision with evacuation of the contents is indicated. A simple form exists, without evidence of being contagious.

Milium resembles the preceding; it is yellowish and elevated, and appears in youth. It should be incised and its contents expressed.

Simple Cysts in the region of the lids should be removed whole.

The eyelids are the seat of a variety of benign and malignant new growths.

Rodent Ulcer is a superficial lesion with a rough uneven floor. Its walls are infiltrated, and as it advances in one direction it heals by cicatrization from the opposite side. It is of slow growth, and years are required to develop it to considerable dimensions. There is no involvement of the regional lymph glands. If not too extensive it should be excised, and the resulting defect can be repaired with skin-grafts. Roentgen rays, with protection to the eyeball, have been effective. It usually attacks elderly people.

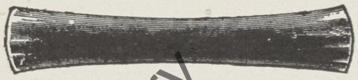


FIG. 7.

Angiomata occur in the lids. The superficial form, *telangiectasis,* shows as a bright red spot in the skin. Deeper ones show as bluish elevations which are compressible. Both kinds should be removed, as some of them tend toward extension.

Blepharitis is an inflammation of the margin of the lids. The mildest form is a local *hyperemia.* Dandruff-like flakes form at the roots of the cilia; when removed, they reveal normal underlying tissue. This form depends upon increased secretions from the conjunctival glands or the glands that open on the lid margin. By placing a Jaeger lid plate (Fig. 7) or similar instrument beneath the lid, and the ulnar edge of a forefinger over the lid at about the supratarsal fold, the contents of the glands can be expressed by rolling the finger to the margin of the lid. The treatment includes the whole extent of

the border. The expressed secretion should be removed. Treatments are repeated once or twice a week.

The placing of the lid plate is not disagreeable after the instillation of a few drops of 4 per cent cocain solution. Where the conjunctival mucous glands are affected a drop of 1 per cent solution of zinc sulphate is instilled three times daily over a period of time.

The *squamous* type is a localized seborrhea. The flakes or scales are difficult to remove, and may require the use of bland soap and water, or peroxid of hydrogen applied with a cotton-wound probe. A reddened and thickened lid border is exposed. Irritation of the sebaceous glands excites an excessive secretion which dries on the lashes when exposed to air. In this form the cilia fall out or are easily removed, but will grow again, as the hair-follicles are not destroyed. Relapses are frequent.

The *ulcerative* type is like an eczema, in that it tends to destroy the hair-follicles. In this form the secretions harden as yellowish crusts, which when removed expose small abscesses. In a developed case these abscesses will be found in a variety of stages, from the beginning of the process to the pitting or scarring which indicates the final stage, with destruction of the follicle and loss of the cilium. The remaining cilia are often found matted together by the secretion. Bleeding may occur when the hairs are removed. All of the follicles may be destroyed; *madarosis* is a total loss of cilia. Because of the inflammation the lid border thickens; connective tissue is proliferated, and by its contraction produces deformities. The margin of the lid may become inverted (*entropion*) and cause the lashes to irritate the conjunctiva and cornea (*trichiasis*) by being turned toward those structures. If the lid border is sufficiently congested or hypertrophied it will droop of its own weight (*tylosis*) and resemble ptosis.

Where the lower lid is affected the processes will be the same, but the effects will differ. By its weight the border will be everted (*ectropion*) from the globe; the punctum does not then occupy its normal position and tears overflow the cheeks. In this deformity, an area of reddened conjunctiva is exposed, and the condition is known as "blear eye." Practically all cases are complicated by a chronic conjunctivitis, and blepharitis is one of the most chronic diseases known in ophthalmology.

The *causes* of the disease are numerous. Some cases are hereditary or familial. Undernourishment, unhygienic living and errors of refraction contribute to its incidence. Irritants as smoke, heat and dust, when continued, may produce it; habitual and prolonged loss of sleep predispose to it. Phlyctenular disease of the conjunctiva and cornea, and some cases of trachoma and chronic conjunctivitis are accompanied by an excessive flow of tears; the

lid margin is moistened continually, an irritation is excited, and blepharitis develops.

Treatment.—The causes should be ascertained and removed, the constitution should be built up, and the patient trained to care for himself. Ointments are the logical bases for medicines. Fats protect the surface from moisture (tears), tend to make the skin pliable, encourage a flow of secretion from the hair-follicles and ducts of the glands, and soften the scales and crusts, which aids in their removal. Lanolin meets the requirements, but is too stiff; the addition of an equal part of petrolatum corrects the stiffness, and the application will remain longer.

In such a base may be incorporated 1 or 2 per cent of yellow oxid of mercury, ammoniated mercury, boric acid, ichthyol, or 1 per cent of salicylic acid. These may be rubbed in with the finger along the roots of the lashes and into the lid margin two or three times a day, and at bedtime. The diseased surfaces must be patiently cleansed with bland neutral soap and water, hydrogen peroxid, warm boric acid or sodium bicarbonate solution, or plain warm water; loose hairs must be removed.

In the ulcerative form, daily search must be made for abscesses which should be opened. Gentle massage over a lid plate is beneficial. Errors of refraction must be corrected. Medical treatment must be continued long after a cure is manifest, because of the tendency to relapses and recurrences. Surgery may be required to correct deformities.

Phthiriasis is a condition in which crab-lice or their nits are found on the cilia. The lashes look dark, as though covered with a fine powder. Unguentum hydrargyri is indicated.

GENERAL SURGICAL PRINCIPLES

The technic for surgical asepsis must conform to that practiced in major surgery. The preparation of the operative field requires special mention. The lashes are trimmed with scissors that have been dipped in sterile water; then the hairs will adhere to the blades. The roots of the lashes are touched with one part U.S.P. tincture of iodin in two parts of 70 per cent alcohol. The operative field and an ample border are painted with the same solution, which is allowed to remain until dry. Where desired, the color can be removed with alcohol or ether. When iodin is objectionable, the field may be prepared with a solution of neutral soap and warm boric acid solution.

In neither method should the fluids be allowed to come into contact with the globe. Sterile petrolatum is introduced into the conjunctival sac and spread over the cornea with the lid.

If a general anesthetic is selected for children or nervous patients, begin with ethyl chlorid or nitrous oxid, and continue with ether vapor by tube under pressure. Then the anesthetist will not discommode the surgeon. Ordinarily, local anesthesia will be selected. Freshly prepared 1 or 2 per cent novocain (procain) solution, containing two or four drops of 1:1,000 adrenalin chlorid solution to each cubic centimeter, will be found effective after a period of fifteen to twenty minutes. All tissues that will be traumatized should be infiltrated, but not to a degree that will distort the cutaneous surface. Consideration for the ultimate cosmetic effect is important.

In preparing to treat abscesses, furuncles and hordeoli, the infiltration is begun at a distance and from several positions, but in the direction of the lesion; the fluid may be gradually forced into the affected area by slowly advancing the needle. Blocking may be effected by injecting the solution at the site of each nerve as it enters the lid area from the orbital margin.

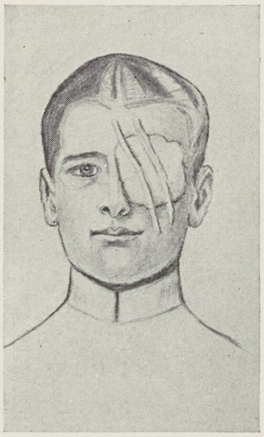


Fig. 8.—Eye Dressing.

Cutting instruments, knives and scissors, must be keen edged, and needles must be very sharp pointed. The skin of the lids is so loosely applied that it easily drags when dull instruments are used. There are a variety of lid clamps on the market, and one of these may be selected to hold the lid while operating; by judicious pressure of the blades the operator can control hemorrhage.

Before beginning the actual operation, a few drops of freshly made 4 per cent cocain solution should be instilled into the conjunctival sac to anesthetize it and the cornea. It is advisable also to instill a drop or two into the fellow eye; this will prevent spasm of the lids in the event of the accidental introduction of any foreign material into that eye.

To protect the cornea on the side undergoing operation, sterile petrolatum may be used; if this is objectionable, the eye may be moistened occasionally with a drop of warm normal salt solution. This protection is important, for during the operation there can be no winking to distribute the tears; cocain loosens the corneal epithelium, and rapid evaporation may produce epithelial desiccation.

Suture materials consist of fine braided or twisted white and black silk for surface, and fine catgut for buried sutures. Twisted black silk offers certain advantages; it is readily found when the suture is to be removed, and it does not seem to irritate tissues unduly. Its disadvantage is its readiness to come untied. It is sometimes hard to draw through tissues. This may be overcome

by winding it loosely on glass spools or rods, and boiling it in a mixture of one part of beeswax and two parts of petrolatum.

For antiseptic dressings, White's ointment usually meets all requirements. This consists of 0.01 gram (1/6 grain) of bichlorid of mercury, 0.054 gram (5/6 grain) of sodium chlorid, enough alcohol to dissolve them, and equal parts of petrolatum and lanolin to make 30 grams (1 ounce); it is a 1:3,000 bichlorid of mercury ointment. This may be deposited in the conjunctival sac and on the external wound. The field is covered with an eye pad. This is made by placing a layer of cotton or cotton wool between layers of gauze, and shaping as desired with scissors.

This pad is placed over the closed lids, and secured by strips of zinc oxid adhesive (Fig. 8). The pressure may be graduated as desired by the thickness of the pad or pads, and the tension put on the skin of the forehead and cheek by the adhesive tapes.

SURGICAL TREATMENT OF FURUNCLES, HORDEOLI AND CHALAZIA

Furuncles and Hordeoli are pyogenic infections. They are identical except in location. Boils appear on the surfaces of the lids, and styes on the borders in connection with the lashes or the ducts of the meibomian glands. Hordeoli may be indirectly due to errors of refraction. In such cases use of the eyes causes a smarting or itching sensation, and the individual rubs the lids. In this way an infection can be carried by the hands.

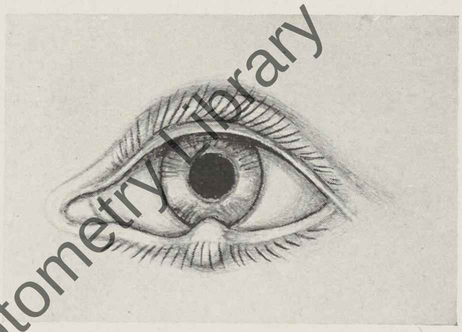


Fig. 9.—Hordeolum Externum.

If either of these processes are seen early, they may be aborted by the application of iced water five minutes out of each waking hour. Two per cent yellow oxid of mercury ointment should be rubbed about the spot at bedtime. Their maturity may be hastened by the use of hot compresses ten minutes out of each hour. To be effective these should be made thick, as by folding a bath towel to the desired size. This is wrung from hot water, applied, and covered with paper, rubber tissue, or oiled silk to retain the moist heat.

When mature, the lesion must be promptly incised and its contents evacuated. Furuncles should be opened by a short incision parallel to the lid border. The contents may be wiped out with a cotton-wound probe, or curetted with a small dull spoon, but without breaking down the defensive wall.

Hordeoli are of two varieties: Externum (Fig. 9) when connected with a hair-follicle, and internum when from an infection of a meibomian gland. The external form may be punctured by a thrust of a narrow thin-bladed cataract knife. Where it is deemed necessary to open a stye above the lash line, a lid clamp is applied and the incision is made parallel to the lid border. The contents should be evacuated in the manner recommended for furuncles.

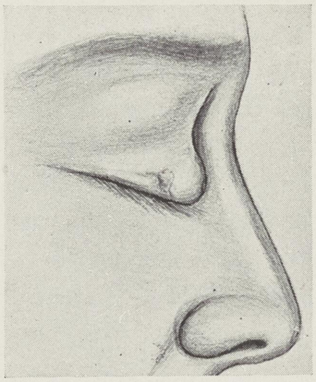


Fig. 10.—Chalazion.

Chalazia are tumors of the lids without typical inflammatory evidence, and always appear within the area of the tarsus (Fig. 10). They are of slow development, painless, and are filled with granulation tissue or the degenerated products of it. The chalazion can grow inwardly toward the conjunctiva, or outwardly toward the skin. In the former instance the conjunctiva beneath the chalazion is eroded, and granulations present through the opening. Before this erosion occurs, the conjunctiva at the point of pressure will have a bluish or yellowish appearance. When this spot is touched with a probe, the membrane is found to be very thin.

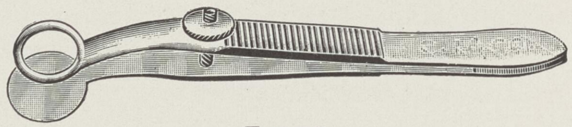


Fig. 11.

As there is no inflammatory zone about the tumor, novocain infiltration may be begun immediately around it. Before making the incision, cocain solution is instilled into the conjunctival sac.

Fig. 12.

The instruments needed are a small scalpel, lid clamp (Fig. 11), serrated curet (Fig. 12) and small sharp curet (Fig. 13). The lid should be grasped with the clamp and everted, with the fenestrated blade on the conjunctival side of and surrounding the tumor. The clamp is held in position by the fingers and *not* by the screw nut. The incision is made *at a right angle* to the lid border, and into the sac. Both curets are employed to clean out the contents and destroy the wall of the cystlike tumor. Hemorrhage follows this procedure.

Remove the clamp and grasp the site of the sac between the finger and thumb. If the sac has not been totally destroyed it will be detected by touch, and further curettement will be necessary. If the sac has been destroyed, continued pressure between the finger and thumb will arrest the hemorrhage, and the space will not fill with clotted blood to the same extent as it would otherwise. By incising at right angles with the lid margin, unaffected meibomian glands are not injured, and the resulting scar comes

into contact with the smallest possible portion of cornea during lid movements. Irritation of the cornea is minimized by this plan.

Where the chalazion grows toward the skin surface, the conjunctiva will show no change; then the tumor should be removed from the outside. The incision should be made parallel with the lid margin in this instance (Fig. 14). The same instruments are used, but the window of the clamp must be turned toward the skin surface.

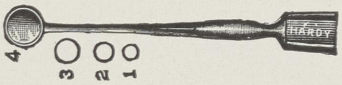


Fig. 13.

A preferable method is to dissect the sac free. The curets are exchanged for a pair of small scissors. The clamp must not touch the globe, and the latter should be protected with petrolatum or White's ointment. The screw nut is tightened enough to check the hemorrhage. An assistant manages the clamp.

The incision is carried through the skin, superficial fascia and the muscle layer, unless the last has been displaced by the growth. It is better to dissect these tissues layer by layer. When the sac is exposed it will present as a bluish bulging rounded mass. With a dissector (Fig. 15) or the closed points of the scissors the sac wall is freed from its attachments. The duct is snipped off. The tumor is lifted out and inspected to see that it is whole. It is difficult to dissect out cleanly, because the walls are easily torn. Two or three silk sutures are needed to close the incision. The clamp should be removed, and the wound grasped between the finger and thumb as before to prevent filling with blood-clot.

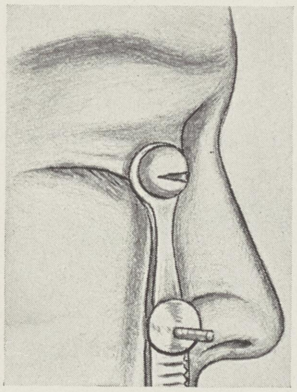


Fig. 14.—Operation for Chalazion.
(After Wheeler.)

Furuncles, hordeoli and chalazia should be dressed, after operation, with White's ointment and eye pads for one day. After the removal of chalazia by dissection the dressings are continued for three or four days; then the sutures are removed.

Abscesses of the Lids are treated like furuncles, but the curet should not be used. Gently cleanse the cavity with cotton mops, irrigate with 1 : 8,000 solution of bichlorid of mercury, and maintain drainage. Due attention to healing is essential, for contraction of the tissues with deformity of the lids is prone to occur, especially if the abscess is large or deep. When this tendency is noted, it is advisable to freshen the lid borders at opposing points and suture them together. The union is not disturbed for several weeks or months, according to the process of contraction. The effect is to immediately protect the cornea from exposure, broaden the scar, and prevent ectropion.

Thiersch's grafts can be applied to the granulating surface of the healing wound. Where the abscess is due to erysipelas, incision, drainage, and gentle irrigation are indicated; suturing of the lid margins should be postponed until the disease is well exhausted; then suturing may be unnecessary.

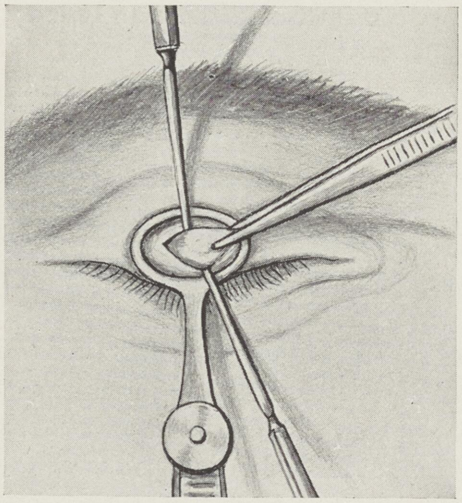


Fig. 15.—Operation for Chalazion. (After Wheeler.)

Epicanthus is a fold of skin over the inner canthus of each eye. When in a child it usually disappears as the bridge of the nose develops. When in an adult, and disfiguring, it can be remedied. By pinching up the skin over the bridge of the nose and noting the effect in exposing the canthi, the area of tissue to be removed can be estimated. Elliptical cutaneous incisions of equal curvature and length are made on each side of the median line of the nose, and the outlined area of skin is excised. The borders outside this area are undermined for a few millimeters, and the gap is closed with interrupted sutures of fine silk or dermol. These are removed after five days.

External epicanthus or blepharophimosis may conceal the outer canthus. A horizontal incision directly outward divides the cutaneous fold into portions corresponding to the upper and lower lids. The angle of the incision is sutured first. Then for each lid the edges of the wound are united with interrupted sutures. The raphé is divided only when indicated.

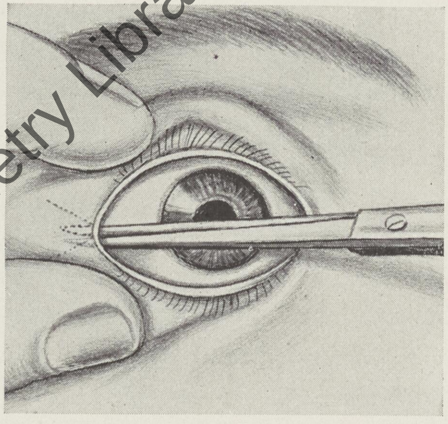


Fig. 16.—Subcutaneous Canthotomy.

Canthotomy and canthoplasty are performed by inserting a thin narrow-bladed knife into the outer angle of the lids, and undermining a triangular area of skin temporally, with the apex of the triangle inward. Each side of the triangle is about 1 centimeter in length. Introduce sharp stout scissors, with one blade under the triangular flap, the other beneath the conjunctival surface of the commissure, and divide the lateral palpebral raphé to the orbital margin (Fig. 16). A canthotomy is for temporary effect; no sutures are used, and the edges of the wound soon reunite.

A canthoplasty is for a permanent effect. A double-armed suture is used; one needle is passed through the conjunctiva near the margin of the orbit, and may include the periosteum, while the other is introduced medial to the first in loose bulbar conjunctiva. The needles are then passed through the skin flap from within outward and the suture is tied over a small roll of gauze or a thin lead plate (Fig. 17). This draws the skin flap down into the wound, and separates the divided tissues. The ends of the suture are passed through two holes in the plate before tying.

Another method is to outline a triangular flap in the form just described, but by incisions through the skin. These are drawn divergently from the canthus the required distances. The skin is undermined from the temporal extremities of the incisions (Fig. 18) to the apex of the triangle at the canthus. The flap is then turned back. The deep tissues are divided as already described.

FIG. 17.

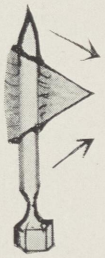


FIG. 18.

The conjunctiva at the lateral fornix is sutured to the skin bordering the denuded area on each side of the deep incision. Another suture is first passed through the bulbar conjunctiva as far laterally as possible, and then through the point of the flap. The flap is drawn deeply between the divided portions of the raphé to permanently separate them. The sutures are removed after five days.

Lagophthalmos designates an inability to close the lids; it is due to protrusion of the globe, paralysis of the orbicularis oculi, symblepharon, or insufficiency of the lids such as might result from the contraction of scar tissue. The cornea is exposed to desiccation and must be protected with sterile petrolatum, a Buller's shield, a bandage or by tarsorrhaphy, as indicated.

Ankyloblepharon is a union of the free borders of the two lids. It is congenital or due to injury or disease. Division of the margins may give a desired effect. Reunion must be prevented by keeping the lid borders separated mechanically, and by the free use of sterile petrolatum until the surfaces are covered with epithelium.

Blepharospasm is either tonic or clonic. The former is due to the presence of a foreign body in the conjunctival sac or on the cornea. Removal of the offending material will relieve it. The clonic type varies in degree. Frequently patients complain of a twitching of the lower lid at the outer canthus. This occurs in cases that have been incompletely refracted, and after prolonged use of the eyes for near work; careful attention to the refraction usually brings relief.

The extreme case is apparently a neurosis, in which the patient experiences periods during which it seems to be impossible to keep the eyes open. Sedatives seldom give relief. Some cases are benefited by one or repeated injections of

1 cubic centimeter of 2 per cent solution of novocain. The needle is entered near the outer canthus and directed first toward the temporal, and then toward the zygomatic branches of the facial nerve; the solution is divided between them. If this has to be repeated several times, a canthotomy or canthoplasty, preferably the latter in bad cases, will be required.

Blepharochalasis occurs from thinning of the skin above the supratarsal fold. The redundancy overhangs the area outlined by the tarsus; it is usually more pronounced laterally, and has been compared to a hound's ear. Enlarged veins often mark the surface. The superfluous skin may be excised.

The first incision is begun not nearer the inner canthus than is judged necessary; it is made 1 or 2 millimeters above and parallel with the supratarsal fold. The skin is divided as far temporally as required. The second incision is begun at the same point, so that the apex of the denuded area is toward the nose. The amount of skin to be removed is estimated, and the position of the second incision is located accordingly. It is better to remove too little than too much, for when the wound is closed there must be no traction on the tarsus to draw the lid away from the eyeball. When necessary, the denuded area can be enlarged with sharp scissors.

Interrupted sutures are introduced to include the superficial fascia at the tarsal margin. The upper lip of the wound will be longer than the lower. By suturing from the medial end, this excess of skin will lie below the eyebrow laterally. It is disposed of by a triangular excision. The apex of this denuded area is directed upward. It is closed with interrupted sutures. Accurate coaptation is a cosmetic requisite.

The incision lines should be covered with White's ointment. Pads are fitted for making moderate pressure on the operated area, and the dressing is continued for eight days. The sutures are then removed.

Ptosis Adiposa has a similar appearance to the preceding. The overhanging skin is thick and sags from its own weight. This condition rarely needs treatment, but when elected, it is carried out on lines similar to those outlined for blepharochalasis.

Solid Edema of the Eyelids presents a thickening which resembles a redundancy. It is largely limited to the lid area (see Fig. 1) and vicinity, especially below. It is a bilateral affection, but may not be symmetrical in occurrence. Some involvement of the regional lymph glands is generally present. It is not a common disease.

Solid edema can usually be divided into three stages: (1) The skin is brawny, red and tender; constitutional reactions are similar to those found in erysipelas and cellulitis, and it can be mistaken for either. (2) The symptoms subside; the skin is dusky and pits easily upon pressure. This stage may pass

to either the first or third. (3) The skin is pale, thickened, stiff and doughy. Remissions and exacerbations are characteristic. The thickening of the tissues is progressive from successive attacks.

The origin of the disease in some cases has been traced to staphylococci in the nose. The treatment consists of cleaning out the source of infection and administering vaccines.

FIG. 19.—COLOBOMA OF THE EYELID. (After Spaeth.)

Coloboma of the lid is a notch in the free border (Fig. 19). These are not common; they vary in size and location, and each one demands a special plan for its correction. In general, the edges of the cleft should be freshened; the conjunctiva is sutured with very fine catgut, and the skin with silk. The two lines of sutures should be planned so that one lies medial to the wound, while the other lies temporal to it. This avoids the deformity due to cicatricial tissue forming through the thickness of the lid.

TRICHIASIS, ENTROPION AND ECTROPION

Distichiasis occurs when the meibomian glands fail to develop and cilia grow in that location. Such hairs turn inward and irritate the conjunctiva or cornea. It is sometimes considered as trichiasis, and the treatment is the same. Structurally, the deformities are distinct.

Trichiasis occurs when the lid is deformed by cicatricial contraction due to injury, blepharitis, severe conjunctivitis, trachoma, or ulceration of the lid. The true lashes are turned toward the eyeball. It is usually combined with entropion. The simplest form of correction consists of transplanting the line of lashes. Anesthesia is effected by novocain in the lid, and cocain to the conjunctiva.

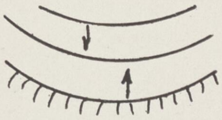


FIG. 20.—INCISIONS.

An incision is made through the skin parallel with the lid margin and 4 or 5 millimeters from it, and equal in length to the line of the lashes. A second incision 3 or 4 millimeters further from the lashes parallels the first, and is 4 or 5 millimeters shorter at each end. These incisions outline a first flap that is undermined for its whole extent. The lid margin is then split along the gray line between the cilia and the ducts of the meibomian glands, in a plane parallel with the lid surfaces, and to the first incision described. This constitutes a second flap (Fig. 20).

Silk sutures are placed in the first flap along the margin nearest the lashes, and this flap is drawn beneath the second. The sutures are then introduced into the margin of the conjunctival surface and tied; this makes a lash-free border for the lid (Fig. 21). The upper margin of the second flap is then sutured to the skin on the undisturbed side of the second incision. The transposed flaps may be sutured together if desired. Ointment is applied and pressure is effected by pads and adhesive tapes. After five to seven days the sutures are removed.

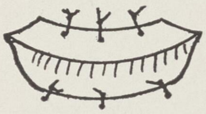


Fig. 21.—Flaps Transposed.

If but a few lashes impinge upon and irritate the eyeball, they may be removed with a cilia forceps (Fig. 22). The follicles are uninjured and the deformity will recur. The follicles can be destroyed by electrolysis. Novocain anesthesia is employed by infiltrating along the border of the lid.

A fine needle is attached to the negative pole of the galvanic current, and the positive pole is applied over the skin of the temple, forehead or neck. The needle is inserted into the hair-follicle and 2 milliamperes of current are used. Fine bubbles of gas indicate loosening of the lash, which can be removed by a gentle pull. The results are usually permanent. If these plans fail, one of the operations for the correction of entropion must be performed. These contemplate a new position for the line of lashes.

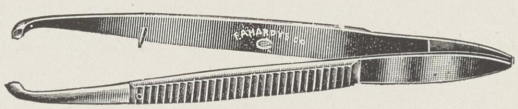


Fig. 22.

Entropion is a rolling in of the free border of the lid. As the lashes are rotated in, the symptoms are those of trichiasis combined with lid deformity. There are two varieties of entropion, spastic and cicatricial. The former is found more particularly in elderly people; there is a loss of orbital fat with spasticity of the orbicularis oculi. The latter form results from disease or injury, more especially from trachoma.

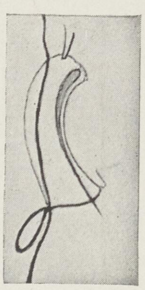


Fig. 23.

Stellwag modified a Snellen suture as follows: Two double-armed sutures are prepared. The lower lid is laid off into thirds horizontally. One pair of needles is passed from the fornix through the entire thickness of the lower lid at the junction of the medial and middle thirds. The needles are reinserted at their points of exit, carried between the tarsus and the orbicularis upward and are brought out in front of the lashes (Fig. 23) where they are tied over metal plates. Similar sutures are used at the junction of the middle and lateral thirds of the lid. The sutures are left in situ for ten to fourteen days, until fibrous tissue forms along them; it is to this tissue that the success of the operation is due.

Arlt's modification of Gaillard's suture is similar in principle. The sutures are double-armed and the lid is laid off into thirds. The needles are entered near the line of lashes and carried beneath the skin to below the lower margin of the orbit (Figs. 24 and 25), where they are brought out and tied tightly enough to produce a slight overcorrection (Fig. 26).

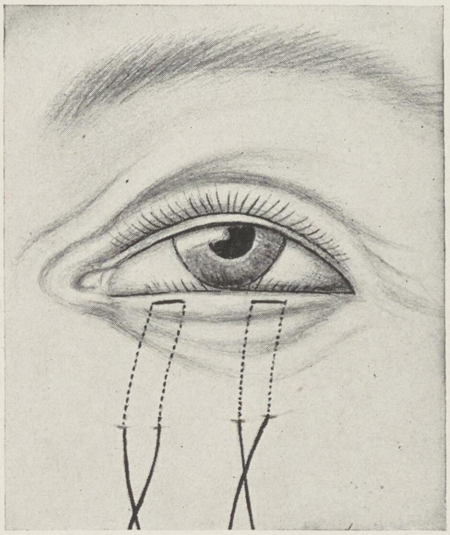


Fig. 24.—Gaillard-Arlt Sutures. (After Fuchs.)

Where the orbital border of the lid is too long, it can be shortened by von Graefe's method. An incision parallels the free border of the lid about 4 millimeters from the line of lashes (Fig. 27). The middle third of this incision forms the base of a triangle of skin to be excised; the excess is estimated by pinching up the redundancy with the fingers (Fig. 28). Beginning at the base line, two incisions are carried downward convergently. The triangular portion of skin is excised. The skin on either side is undermined, until the flaps can be approximated and sutured in a vertical line. No sutures are required for the horizontal incision. The sutures are removed on the fifth day.

An alternate method is to pinch up a horizontal fold of skin along the orbital margin and excise it. The skin on the lid is then undermined and brought down to approximate the line of the lower incision. Sutures are introduced into the margin of the skin of the lid from without inward, a cross-bight is taken in the fascia beneath the lower margin, and the sutures are brought out through the skin below and tied over plates (Fig. 29). Mild overcorrection is desirable in all these methods, especially for the spastic forms where the causative factor is usually progressive.

Fig. 25.

Fig. 26.

Another operation for the same purpose is the galvanocautery puncture of Ziegler. The lid is anesthetized, and protected by a lid plate on its conjunctival surface. The lashes are rotated outward by traction on the skin. Punctures extending well into the tarsus are placed about 3 millimeters apart, and about the same distance from the line of the lashes. The success of this procedure depends upon the formation of scar tissue (Fig. 30).

The foregoing measures are principally applicable to the lower lid, which

is the usual member involved in spastic entropion. For the upper lid, the method given for trichiasis may be used to transplant the lashes to a higher level. For cases with little deformity, a horizontal fold of skin from above the

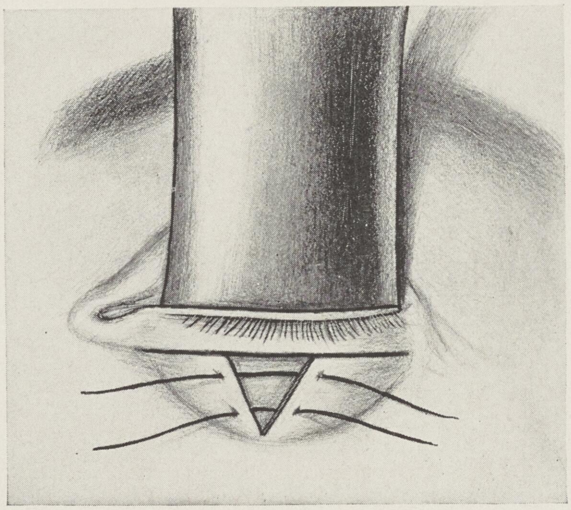


Fig. 27.—Graefe's Method.

tarsus may be excised as outlined for the lower lid (see Fig. 29), or the galvanocautery punctures may be made.

The Hotz operation is performed by making a cutaneous incision from one

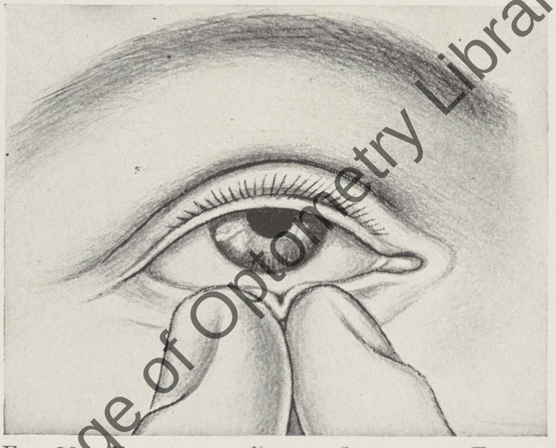


Fig. 28.—Estimating Excess Length of Eyelid.

end of the tarsus to the other along its upper margin. The fibers of the orbicularis oculi are removed for a breadth of 3 or 4 millimeters to expose the tarsus. If the latter is not flexible, it is made so by shaving thin slices from its anterior surface. Three or four sutures are required. Each is placed by

entering the needle through the skin of the upper margin of the wound from without in; a cross bight is taken in the upper border of the tarsus, the lashes

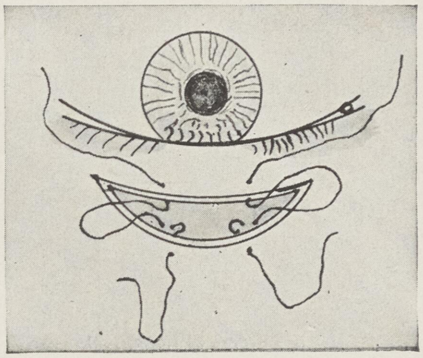


Fig. 29.—Showing Cross Bight.

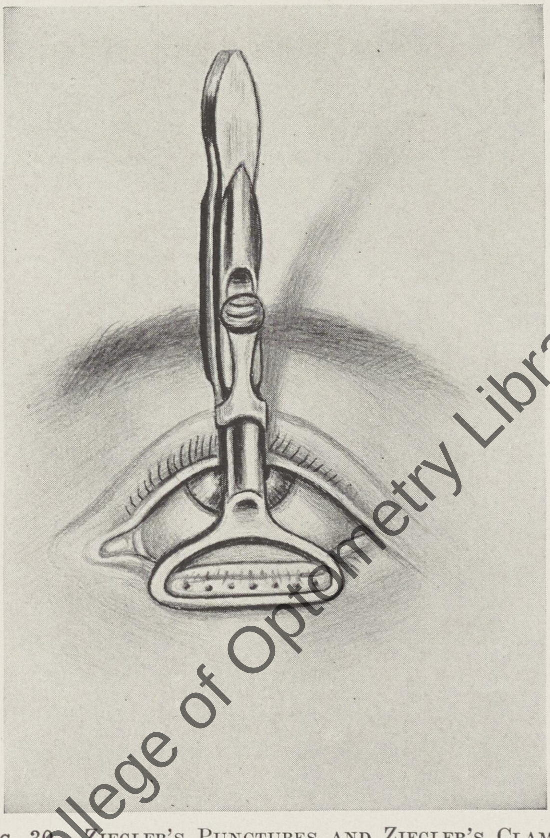


Fig. 30.—Ziegler's Punctures and Ziegler's Clamp.

are adjusted as desired, and the suture is brought through the skin of the lower margin from within out. The sutures are tied over metal plates.

The effect is emphasized where the free ends of the sutures are secured

above the brow by adhesive tapes. The sutures should be left in place for a week, when the supporting strands to the brow are cut. The wound sutures are removed after four to six days more.

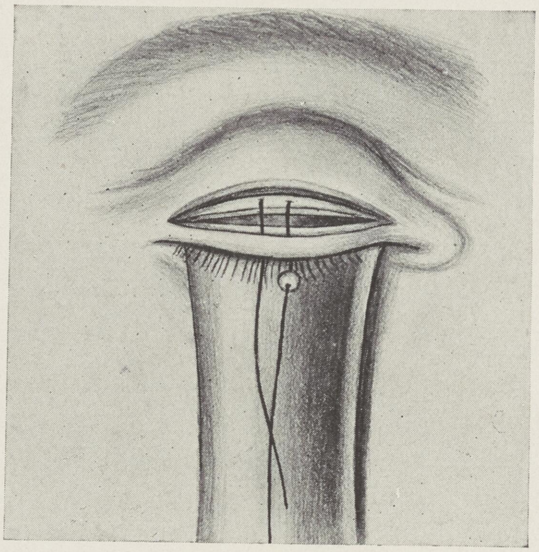


FIG. 31.—SNELLEN-PANAS OPERATION.

In Panas' modification of Snellen's operation an incision is made through the skin equal in length and parallel with the line of lashes, and 2 or 3 millimeters above that line (Fig. 31). The skin flaps should be dissected up to expose the tarsus. The tarsus is divided in its entire thickness exactly in line with the original incision (Fig. 32). Double-armed sutures are used; one needle is passed through the upper margin of the tarsus, and then both needles on each suture are passed beneath the skin and through the free border of the lid behind the lashes (Fig. 33). The desired amount of correction is estimated by traction on the sutures.

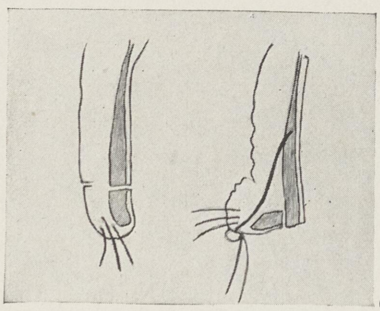


FIG. 32. FIG. 33

Another modification of Snellen's operation is to expose the tarsus as before, but by removing a strip of the muscle; a horizontal wedge is cut from the tarsus down to but not including the conjunctiva. Three sutures are used. Each passes through the upper margin of the tarsal groove just formed, and finally through the muscle and skin of the lower margin of the incision (Fig. 34). The sutures are to remain for a week, during which time the eye is protected by a bandage. The preceding operations are for trichiasis and entropion that affect the central portions of the lids.

The Spencer Watson operation is indicated when either extremity of the lid, and not the midportion, is affected. This is performed by outlining a slender, tapering, pedunculated flap, which includes the offending lashes, with its base toward the nearer canthus; a second flap is formed beside the first but in the reversed direction, so that when the free ends are lifted their bases will lie at opposite ends of the denuded area (Figs. 35 and 36). The flaps are transposed and sutured into place; the sutures are removed in three or four days.

Tarsectomy.—Deformity of the tarsus is due to the effects of trachoma, burns or lacerations. Entropion is usually present. Tarsectomy may be required. An irritable eye demands general anesthesia, otherwise a local anes-

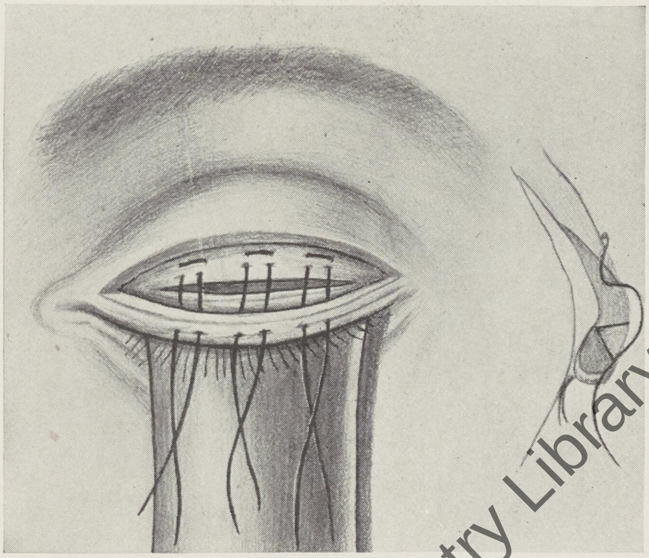


Fig. 34.—Snellen Operation Modified.
Insert shows suture in cross section.

thetic is indicated. The infiltration of novocain must include the tarsal tissue and the injection should be made from both the cutaneous and conjunctival surfaces.

A lid clamp will be needed, and the Ehrhardt is the one of choice for this operation. The lid is raised from the globe and grasped by the clamp, plate side to the skin. The lid is then everted. The conjunctiva between the orifices of the meibomian glands and the ciliary border of the tarsus is caught by the serrated blade of the clamp.

The first incision is made along the whole extent of the ciliary border of the tarsus to the muscle tissue. The incision is extended around the orbital border of the tarsus before the dissection, or the tarsus may be dissected from

the muscle before it is detached above. The tarsal conjunctiva is removed with the tarsus.

If the upper border of the tarsal plate is not deformed it may be preserved, for this portion bears the insertions of fibers from the levator palpebræ

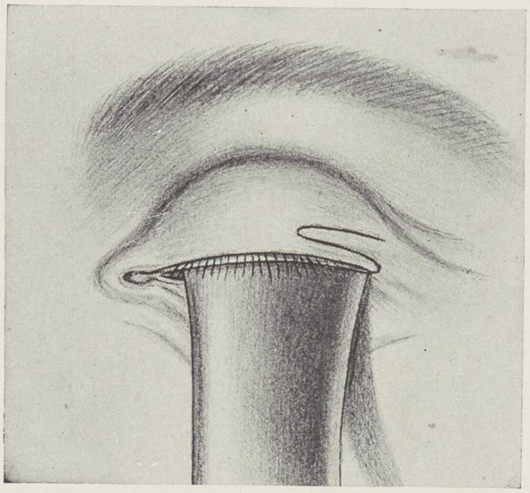


Fig. 35.—Spencer Watson Operation.
(After Meller.)

superioris, and of all the plain muscle-fibers. The conjunctiva is undermined in the superior fornix, if required, so that the conjunctival flap can be brought

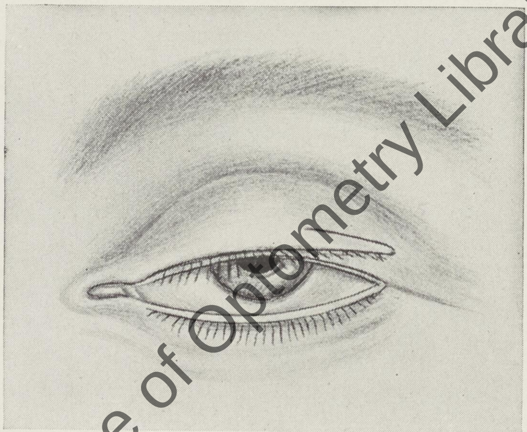


Fig. 36.
(After Meller.)

down to the ciliary margin. It is important that the edges of the flaps be brought together neatly, and then sutured with fine silk. The latter may be removed in five to seven days. This is essentially the operation practiced by Fox of Philadelphia in treating certain types of old trachoma.

These cases are often complicated by ptosis or tylosis. When such complications exist, three double-armed silk sutures are employed by Wheeler of New York. The free margin of the lid is divided into fourths, and a suture is used at the junction of each of the fourths.

While the clamp is in place, one of the needles of each pair is passed through the conjunctiva and the insertion of the levator palpebræ superioris, or the rim

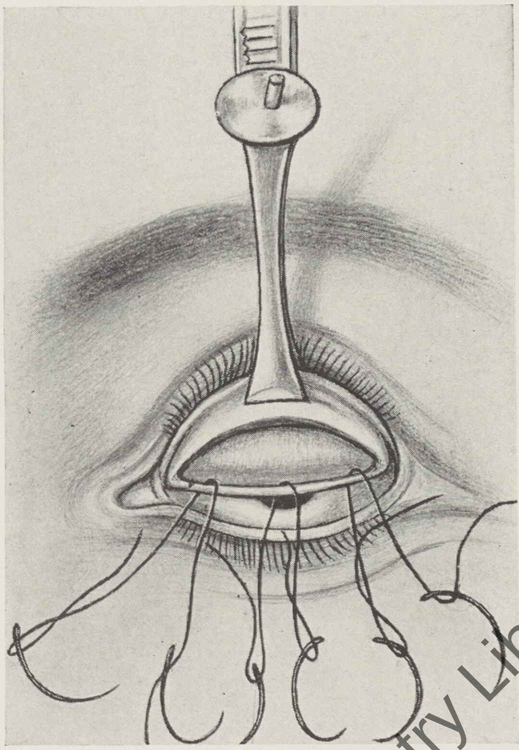


Fig. 37.—Tarsectomy Sutures.
(After Wheeler.)

of tarsus if that has been preserved (Fig. 37). The clamp is removed, and the same needle is passed through the muscle and skin of the lid in the line of the primary incision, while the other needle is passed through all layers of the lid between the line of the incision and the orifices of the meibomian glands (Fig. 38).

A rim of tarsus may be left next the lid border, and then the second needles are passed through this rim. The effect is to raise the lid margin toward the insertion of the levator, and so gain the advantage of that muscle in overcoming the ptosis. The sutures are tied over metal plates or gauze rolls. Dressings can be discontinued after two or three days, and the sutures are removed in six days.

The advantage of this operation in trachoma is that it removes the diseased tissue which produces most damage to the cornea. Partial resections of a deformed tarsus may be performed.

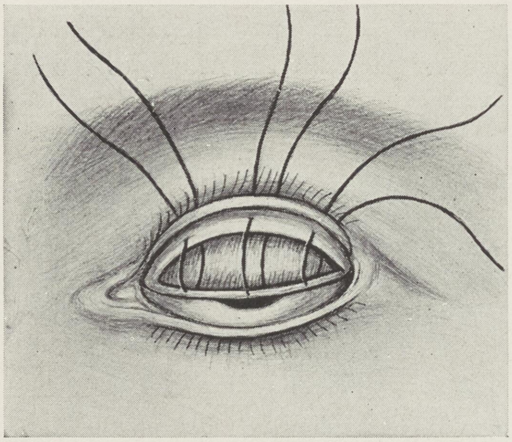


Fig. 38.
(After Wheeler.)

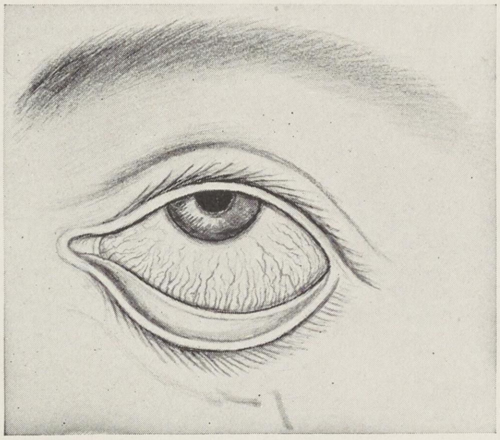


Fig. 39.—Ectropion and Engorgement of Conjunctival Vessels.
(After Dalrymple.)

Ectropion is a rolling out, or eversion of the margin of the lid, with exposure of the conjunctiva and a retraction of the punctum from the lacus lacrimalis (Fig. 39). There are two principal forms: (1) Acute, *spastic* or muscular, and (2) chronic, *cicatricial* or organic. The former is sometimes a feature of blepharospasm. The latter may arise from injuries, deforming diseases, paralysis of the facial nerve, and is occasionally an evidence of senile relaxation (Fig. 40). In cases of blepharospasm with ectropion, the treatment of the former is indicated, with occlusion of the eye for twenty-four hours.

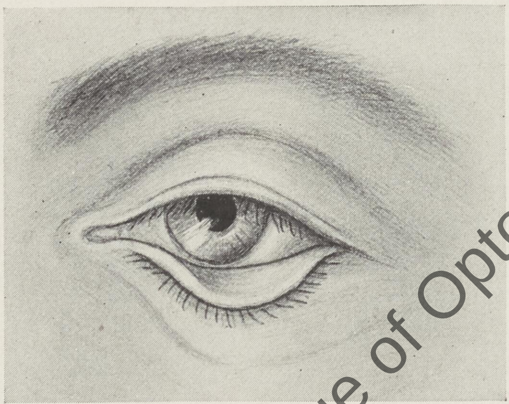


Fig. 40.—Senile Ectropion.

Snellen sutures are employed as follows: Double-armed sutures are inserted into the most prominent part of the ectropion, passed deeply through the tissues, and brought out near the orbital margin (Fig. 41). Two or three such may be used if needed. The effect of tightening the sutures is to draw the lashes toward the globe and replace the lid (Fig. 42). Before tying the sutures the traction effect is

to be noted. Some overcorrection is desirable. The sutures should be tied over metal plates and left in place for ten to fourteen days. A snugly fitting pad will assist in holding the lid in its restored position.

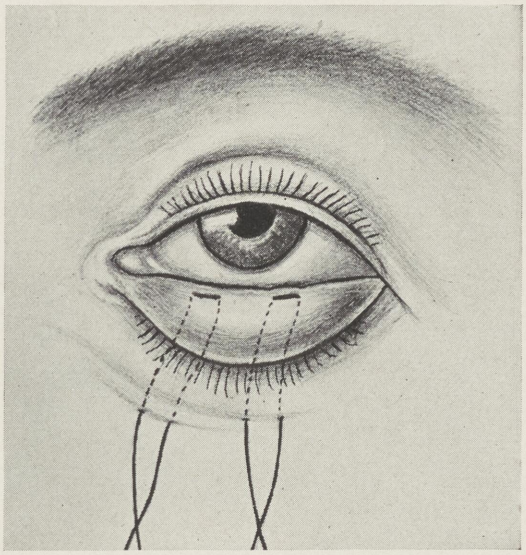


FIG. 41.—SNELLEN'S SUTURES.

Ziegler's galvanocautery punctures are used in the same way as for entropion, except that the punctures are made from the conjunctival surface. They extend well into but not through the tarsus. A lid clamp, with its plate

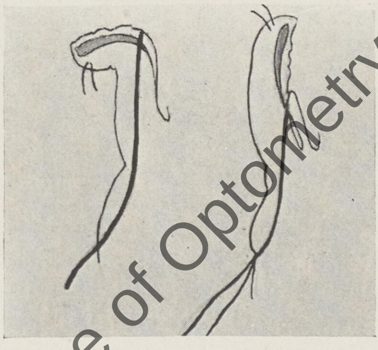


FIG. 42.

surface next the skin (Fig. 43), is used instead of the spatula in this operation.

Some cases of ectropion exist because the free border of the lid is too long. One modification of the Kuhnt operation is performed as follows: The amount of tarsus to be excised is estimated by pinching the lid horizontally between the thumb and finger (see Fig. 28). An incision in the gray line of the lid margin

extends from the medial end of the section of tarsus to be excised, outward to the lateral canthus (Fig. 44). The depth of the incision is equal to the width of the tarsus.

A V-shaped section is cut from the tarsus (Fig. 45). When the edges of the wound are approximated, the lid border should lie naturally against the globe; the gap in the tarsus can be enlarged if necessary. Continuing the curve of the lid border at its temporal end, a cutaneous incision is made laterally from the canthus. This incision exceeds the length of the excised portion of tarsus. From its extremities converging incisions are made downward toward

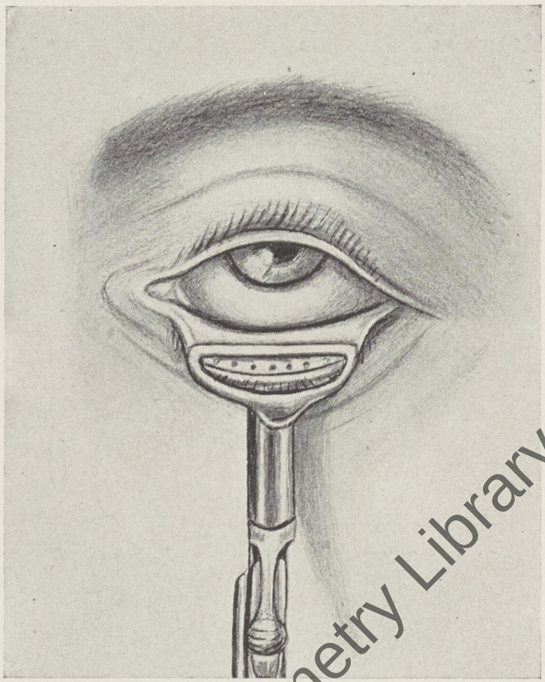


Fig. 43.—Ziegler's Punctures.

the zygoma in the form of a triangle. This triangle of skin is excised, and the musculocutaneous lid flap is drawn temporally to cover the denuded area.

The edges of the divided tarsus are united by two or more sutures extending through both conjunctiva and tarsal plate (Fig. 46). After destroying the follicles of the lashes that would lie beyond the canthus, by trimming them with flat scissors, the lid flap is sutured to the skin at the upper and temporal margins of the denuded area beyond the lateral canthus. Finally, a suture is passed through the margin of the split lid midway between the tarsal repair and the lateral canthus (Fig. 47). Dressings are applied. The sutures are removed in five to seven days.

The Wharton Jones "V-Y" operation is of value where the ectropion is due to a small area of cicatricial tissue in the lower lid near its center. Below

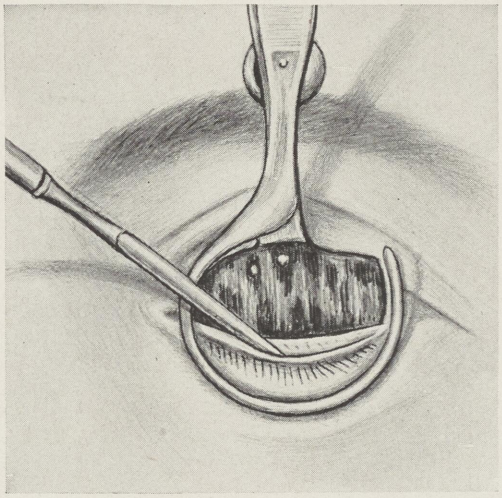


FIG. 44.—KUHNT-DIEFFENBACH OPERATION; KNAPP'S FORCEPS. (After Wheeler.)

the most prominent part of the ectropion two cutaneous incisions are converged downward (Fig. 48). This flap is undermined, and the skin outside the lines

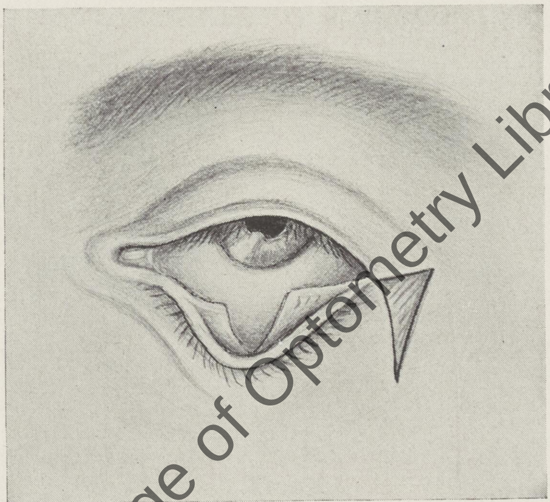


FIG. 45.

of incision is likewise undermined so that it can be drawn to the midline of the denuded area. It is then sutured as far as required to raise the lid border to its normal position. The original incisions are sutured in their new positions (Fig. 49).

Where the ectropion is the result of scar formation, the cicatricial tissue should be dissected away, and skin-grafts used to repair the defect. This is called blepharoplasty. About all methods of grafting are employed. If the area is not too large, a free graft can be taken from another lid in order to get

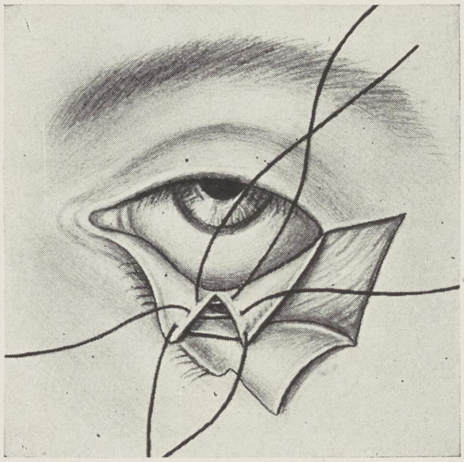


Fig. 46.

an identical quality of skin. Such grafts need be but very little larger than the area to be covered. Skin from the arm or thigh will contract, and the graft must be a fourth to a third larger than the area to be covered.

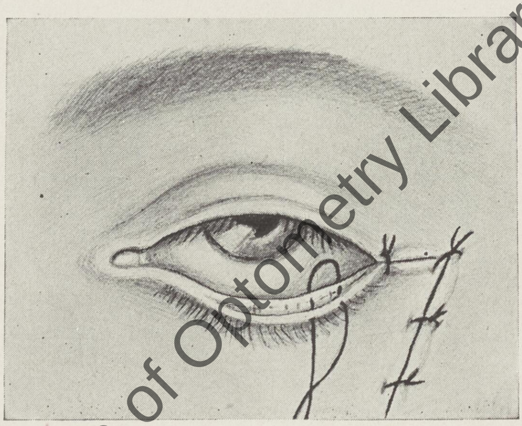


Fig. 47.

Pedunculated grafts may be cut from one lid to repair another. This variety of graft may also be cut from the forehead, the angle of the nose and cheek, or from the temple or face, depending on the proximity of the denuded area to be covered. Such grafts must be oversize. For successful grafting, subcutaneous cicatricial tissue and bands must be excised or divided, the lid mar-

gins must be temporarily united by a tarsorrhaphy, the graft must be fixed at its margins with interrupted sutures and applied to its bed with a judicious degree of pressure, and both eyes must be bandaged long enough for the graft to take.

Pads made of cotton wool are suitable to maintain pressure over the grafted area. They are built up to a required thickness, and secured by an elastic bandage. The tarsorrhaphy should be retained for several months, or until there is no danger of deformity resulting from delayed contraction, which would defeat the purposes of the operation.

A third form of ectropion is called *paralytic*. It is symptomatic, and is

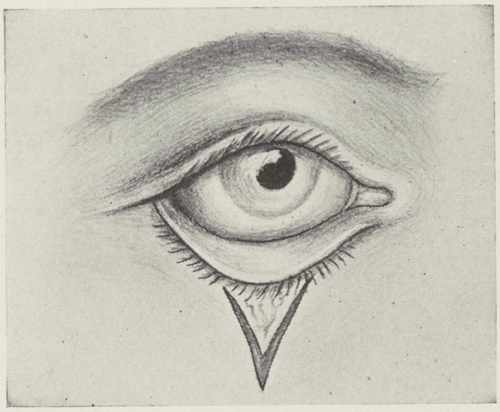


Fig. 48.—Wharton Jones Operation.

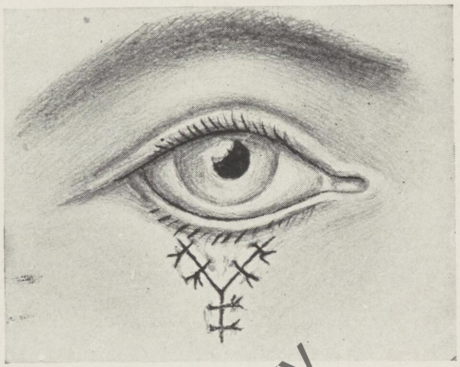


Fig. 49.—Operation Completed.

found in cases of paralysis of the facial nerve. The ectropion affects the medial half of the lower lid. The punctum is retracted from the eyeball, and epiphora results. Where the lower lid cannot be closed, the cornea is exposed; its epithelium dries and infection is invited. Keratitis and corneal ulcers may lead to loss of the eye.

Mild cases are bandaged at night; the more serious are kept bandaged constantly, and the severe ones require a partial tarsorrhaphy. The cause of the paralysis requires treatment.

PTOSIS

Ptosis or blepharoptosis is a drooping of the upper lid. Unilateral cases are generally due to paralysis of the levator muscle. Contraction of the frontalis, with raising of the eyebrows and horizontal wrinkling of the skin of the forehead, and tilting of the head backward with turning of the eyes downward are characteristic features of the bilateral form of the disease; it is congenital or acquired.

Pseudoptosis includes ptosis adiposa, the thickened and sagging lid of the old trachoma case in which there is some spasm of the orbicularis, lid tumor, edema, emphysema, blepharochalasis, and retraction of the globe with loss of support to the lid. In these the upper lid can be partially elevated without the help of the frontalis. An apparent ptosis due to paralysis of the sympathetic nerve is accompanied by contracted pupil and retraction of the globe (Horner's syndrome).

An acquired ptosis is often associated with paralysis of the superior rectus or other extra-ocular muscles. The paralyzed lid can be held open with the fingers while tests are made for deviation, defective rotations and diplopia. Where the superior rectus also is paralyzed, the cornea is not completely rotated upward beneath the eyelid during sleep. If the ptosis is corrected the cornea will be partially exposed and subjected to desiccation and infection.

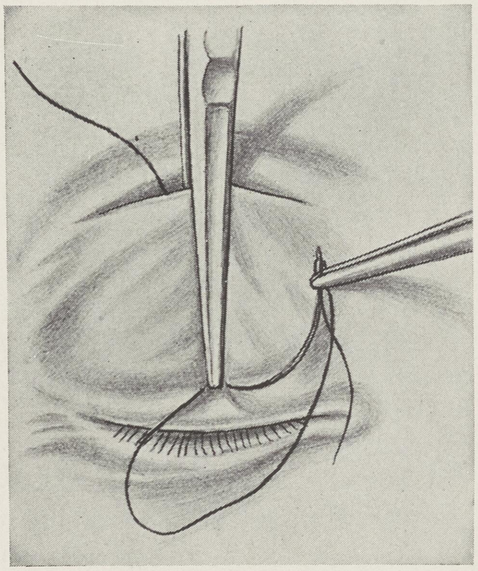


Fig. 50.—Hess Operation.
(After Meller.)

The levator may be only partially paralyzed. The patient is directed to close his eyes and relax the frontalis. The eyebrows are fixed by pressure of the thumbs so that the frontalis cannot contract, and the patient is directed to open his eyes. An upward movement of the upper lid indicates some action of the levator. Where the frontalis has power to lift the lids, an operation is indicated.

Treatment must be directed to the cause in the acquired forms. In the congenital types an appropriate operation must be performed. Motais transplanted the healthy superior rectus to the insertion of the levator; this interferes with the function of the former muscle and diplopia is apt to result. Eversbush transfixed the belly of the levator in three sections, dissected its insertion from the tarsus, excised a bundle of orbicularis fibers, and attached the levator lower down on the tarsus.

Pagenstecher's sutures are employed in moderate degrees of ptosis. Some operators use wire sutures as they are to remain for several weeks. The lid is divided into thirds, and a double-armed suture is inserted under the skin at each of the junctions of the thirds, parallel with the lid margin for 5 or 6 millimeters, and 3 or 4 millimeters above it. The needles are reinserted at

the exits of this horizontal suture, and are carried directly upward subcutaneously to a point some millimeters above the eyebrow. Traction is made to attain an overcorrection, and the sutures are tied over metal plates.

Hess's operation is used in severe grades of ptosis. It is suitable to bilateral cases, because the effects are controllable and can be made equal for the two sides. A horizontal incision is made in the shaved eyebrow for a distance equal

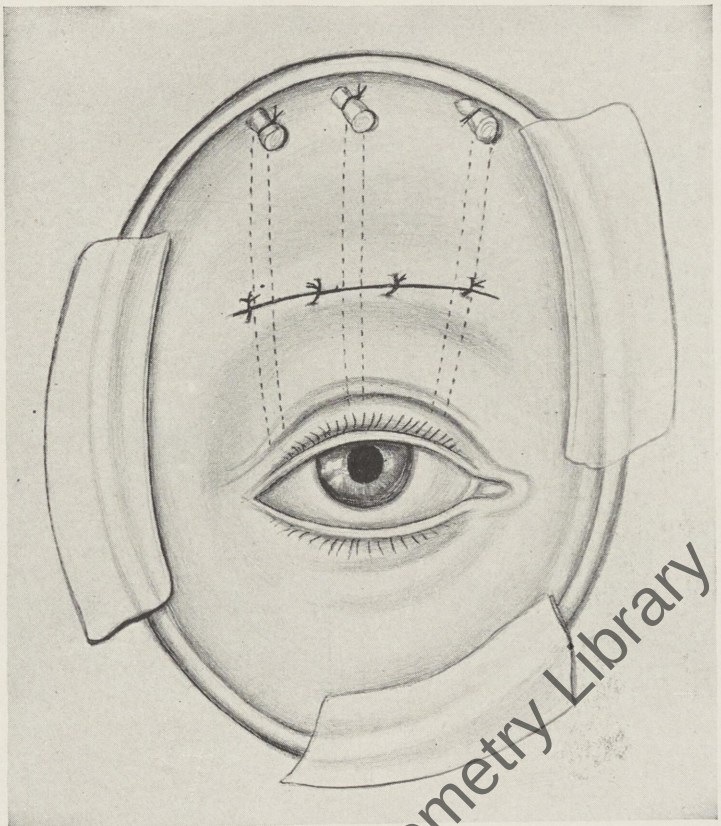


FIG. 51.—HESS OPERATION AND BULLER'S SHIELD.
(After Meller.)

to the length of the palpebral aperture. The skin is dissected from the underlying muscle down to near the lid margin, in the form of a four-cornered pocket.

Three double-armed sutures of strong silk are inserted at the junctions of the fourths near the bottom of the pocket. The intention is to make a new supratarsal fold which should be planned to correspond with the position of the fold in the fellow eyelid. One blade of a fixation forceps is inside, and one outside the pocket to insure accuracy in placing the sutures (Fig. 50).

The needles of each suture are inserted 3 or 4 millimeters apart and are brought out of the pocket above. The middle pair of needles is then passed

beneath the skin at the line of the incision and deeply toward the periosteum behind the eyebrow, and is brought out 1 or 2 centimeters above it. The lateral pair of needles is similarly passed; the medial pair is inclined somewhat toward the median line of the forehead. Traction is made for overcorrection, and the sutures are tied over plates.

Some operators tie the sutures in bowknots, so that additional traction can be utilized, when desired, by shortening the threads. A judicious primary overcorrection should make this unnecessary. The incision is closed with several fine silk sutures.

The dissection of the pocket produces two raw surfaces. Scar tissue forms along the sutures. The success of the operation depends on the adhesion of these surfaces and the support of the cicatricial bands. Overcorrection results in temporary inability to close the lids. A celluloid or glass Buller's shield applied almost air-tight (Meller) will protect the cornea (Fig. 51). The shield is removed temporarily when it is seen that the eye needs cleansing. The skin sutures are removed in three to five days, but the suspension sutures are retained for two or three weeks.

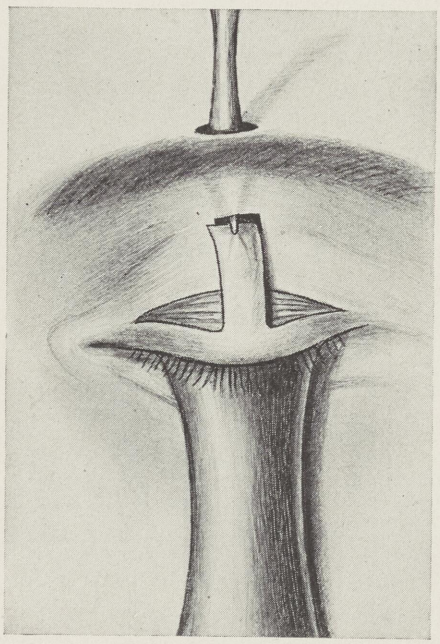


Fig. 52.—Tansley-Hunt Operation.
(After Wheeler.)

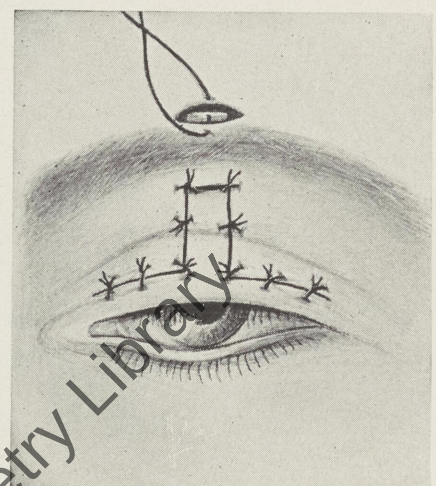


Fig. 53.—Sutures Tied.
(After Wheeler.)

The Tansley-Hunt operation offers assurance of success in severe grades of ptosis, although some puckering of the skin attends healing. An incision immediately below the eyebrow and parallel with it, and 6 millimeters long is made across the vertical midline of the lid. From each end of this incision another is made directly downward toward the lid margin and to within 4 or 5 milli-

meters of it. These incisions extend through the skin and orbicularis, and outline a flap which is dissected from the underlying tarsus.

From each of the parallel vertical incisions others follow the supratarsal fold to both the lateral and the medial extremities of the tarsus, while others extend from the extremities of the last two incisions to the base of the flap (Fig. 52). The skin and muscle are excised from the two triangular areas.

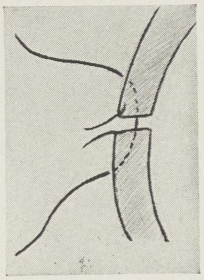


Fig. 54.
(After Parsons.)

A narrow-bladed knife is inserted from below upward into the first incision, and is passed behind the eyebrow to emerge above it. The incision above the brow is made longer than the one below it. The epithelium is scraped from the rectangular flap to freshen that surface. A suture is passed through the free end of the flap, then behind the brow, and out through the last incision. The flap is pulled into place by this suture or with forceps. When slight overcorrection is attained, the flap is secured to the last incision by a suture at each corner (Fig. 53). The excess length is cut off smooth with the surface of the skin of the forehead. The incisions in the lid are closed with interrupted sutures.

Both eyes are bandaged for three or four days. The sutures are removed at the end of a week. The cosmetic results will be acceptable eventually. Where the ptosis is bilateral, both sides should be operated on at the same time.

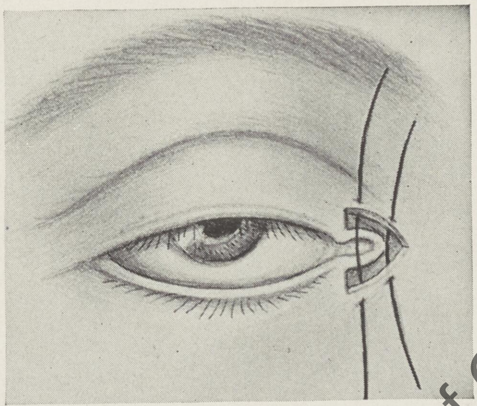


Fig. 55.—Medial Tarsorrhaphy.

Tarsorrhaphy is employed to protect the cornea in cases of lagophthalmos. After freshening corresponding sections of the opposing borders of the lids, they are united at intervals along their entire extent (Fig. 54) or as far as required.

A medial tarsorrhaphy is performed by excising a narrow strip of skin from the vicinity of one punctum to that of the other, by following around but not invading the canthus (Fig. 55). This leaves a denuded area of horseshoe shape, and the upper portion is to be sutured to the lower without touching the canaliculi. When no longer needed, the adhesion can be separated and the canthus will be intact. A lateral tarsorrhaphy is best done by the method devised by Fuchs. The desired amount to be closed is estimated (Meller states that 8 millimeters is the

limit), and each lid is split along the gray line for the desired distance, and 4 or 5 millimeters deep. A lid plate is used to protect the globe. Cutaneous incisions 3 millimeters long are made in each lid at right angles to the lid borders and equally distant from the lateral angle.

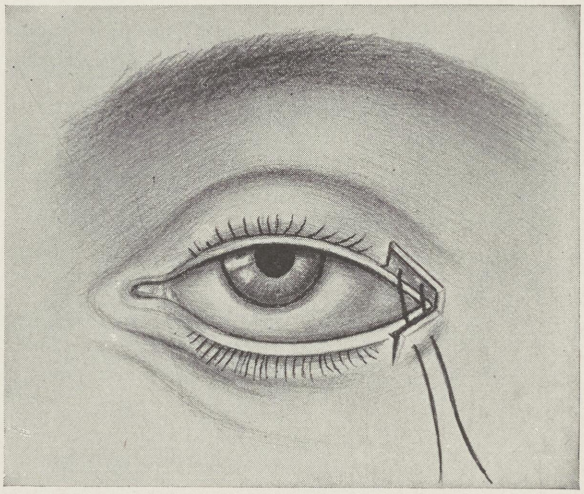


Fig. 56.—Fuchs' Lateral Tarsorrhaphy. (After Meller.)

A rectangular strip of skin 2 or 3 millimeters wide is then excised from the split margin of the upper lid from the last incision to the angle (Fig. 56); this removes the lashes. The flap formed in the lower lid is turned outward and the follicles of the lashes are trimmed out with scissors applied flatly; the cilia will fall out later.

Both needles of a double-armed suture are passed from the conjunctival surface of the upper lid through the bed formed for the reception of the flap from the lower lid, which they also pass through (Fig. 57). The flap is adjusted and the suture is tied

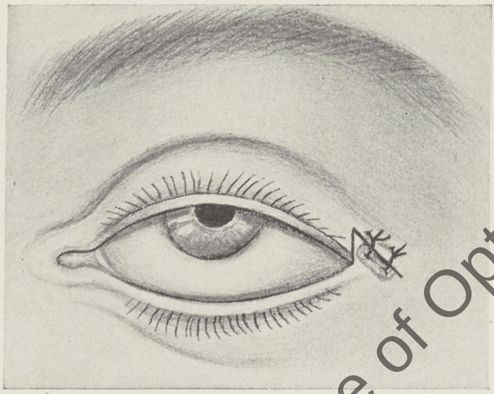


Fig. 57.—Shows Lead Plate as in Fig. 56, and Sutures Tied as in Fig. 58. (After Meller.)

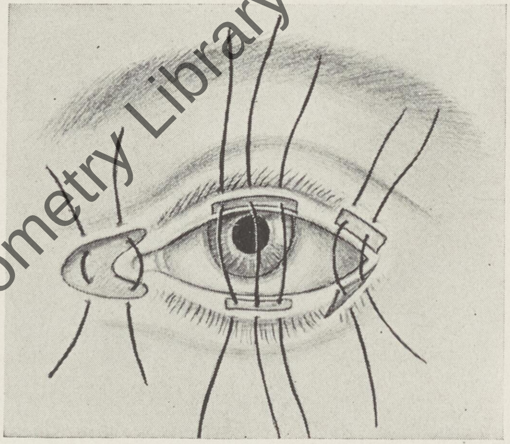


Fig. 58.—Arrangement of Sutures. (After Parsons.)

over a metal plate. Fine sutures unite the skin edges. A dressing is worn until the third or fourth day, when the sutures are removed. This operation aims at permanency; it is employed in incurable facial paralysis where the

cornea is constantly exposed to the air and is in danger from "keratitis e lagophthalmo." Other operations are shown in Figure 58.

Cases of expected short duration can be protected by one of the simpler operations, or by wearing a bandage or a Buller's shield.

INJURIES OF THE LIDS

Injuries of the lids include contusions, lacerations and burns. The structure of the lids permits early and marked edema. Ecchymoses frequently accompany the edemas. They may be due to direct injury of the lids, or to injuries of the contents or walls of the orbit with infiltrations of serum or extravasations of blood. The treatment depends upon the cause. In simple edema with ecchymosis or a "black eye," cold compresses are indicated early; no treatment is effective in the late stages, although potassium iodid internally may assist in clearing the tissues of the extravasated fluid.

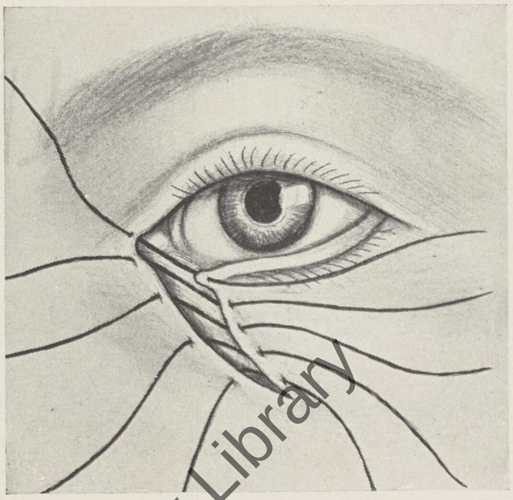


Fig. 59.—Sutures for Repair of Lacerated Eyelid. (After Wheeler.)

Emphysema results from air gaining access to the lid tissues through a perforating wound of the lacrimal sac, or from injury to the lamina papyracea of the ethmoid bone; the inflation does not occur without force, such as blowing the nose. The first instance is one of pure lid emphysema, while the second is an extension of orbital emphysema. The latter is restrained by the orbital septum; when this yields, air enters beneath the skin of the lids. In such a case the inflation extends to the margins of the lids; in orbital emphysema it extends only to the orbital border of the tarsus.

Treatment.—A pressure bandage assists in expelling the air, and the patient must refrain from blowing the nose or sneezing. Attention is given the cause. The emphysema, of itself, is not especially injurious.

Lacerations may include a part or the whole thickness of the lid. Such injuries demand the usual antiseptic treatment. Where the wound parallels the direction of the fibers of the orbicularis, very little if any deformity is apt to result. If the muscle-fibers are broken, the wound has a tendency to gape regardless of its direction. Such wounds require careful suturing. Where the

laceration extends through the border of the lid, special attention must be given to prevent sagging. The conjunctiva and skin should be sutured separately.

Should the wound extend from the inner canthus down and out, the upper inner angle of the outer flap must be overcorrected up and in (Fig. 59). This is accomplished by inserting the sutures at a higher level on the nasal side of the laceration than on the temporal; an additional traction or tension suture may be anchored above to the skin of the nose. Moderate pressure is applied in dressing. The sutures are removed in a week.

Burns are from hot fluids and metal, or caustic acids or alkalies, and require the usual surgical treatment employed for similar injuries elsewhere. When deep, the treatment will be the same as for abscesses and ulcers of the lids.

Foreign Bodies in the lids, if they can be palpated, are removed by cutting down upon them and withdrawing them with forceps. The wound is to be treated surgically.

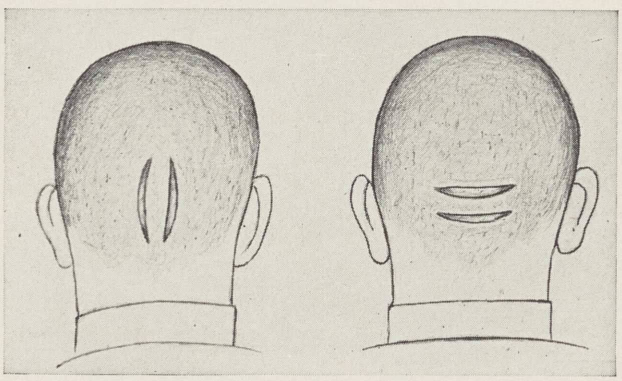


Fig. 60.—Scalp Grafts for Eyelashes. (Partly after Spaeth.)

Madarosis. — Lashes, when destroyed, may be replaced by suitable grafts taken from the upper or lower border of either eyebrow, preferably from the nasal end, or by narrow vertical grafts for the upper lid, horizontal for the lower, cut from the occipital scalp (Fig. 60). The grafts must be set so that the lashes are inclined outwardly for the upper lid and downwardly for the lower.

The bed for the graft is formed by splitting the border of the lid; the graft is set in place level with the lid border, and the lids are immobilized by suturing them together from their skin surfaces. Moderate pressure is applied for a week or more. The tension sutures are to be removed when the grafts are well set and look healthy. The new lashes require careful training to avoid trichiasis.

DISEASES OF THE TARSUS

Tarsitis or inflammation of the tarsal plates is primary or secondary. It is characterized by thickening of the lid and deformity. Complicating tylosis or ptosis of the upper lid and ectropion of the lower are probably due to the weight of the thickened tissues. Sloughing may occur.

Primary Tarsitis is alleged to be syphilitic gumma, since it is occasionally found during the tertiary stage of syphilis. Two forms have been described; in one there is a diffuse gummatous inflammatory infiltration, while in the other there is a circumscribed noninflammatory gummatous thickening. The latter is the more common. The thickened plate can be palpated beneath the skin; it is not particularly tender or painful. The lid is sometimes so thick and swollen that it cannot be everted. Hyaline and other changes are of such a nature that when pieces of tissue are desired for histologic study they can be excised without bleeding.

Single or multiple hard nodules deform the lid; they are not movable on the tarsus. The nodules may undergo hyaline degeneration, or they may break down and ulcerate. Neighboring lymph glands are occasionally swollen. The skin and conjunctiva may or may not be invaded. Salvarsan has given good results in this form.

Acute Tarsitis occurs with signs of conjunctivitis and blepharitis, usually in tuberculous subjects. It is probably due to an extension of a tuberculous process from the lids. The local treatment is yellow oxid of mercury ointment for the blepharitis. General treatment is indicated for the tuberculous condition.

Secondary Tarsitis is mostly found as a complication in trachoma. The tarsus is contracted, shortened and deformed. Contractions, scars, and other evidences of trachoma will be present. Tarsectomy is indicated.

A necrotic form of tarsitis or a tarsadenitis has been reported. It may be a chronic suppurative infection of the meibomian glands in which the tarsus is secondarily broken down.

Amyloid Degeneration of the tarsus has been reported in connection with severe cases of trachoma. It might be confused with sarcoma and carcinoma. The diagnosis is made in the laboratory. No effective treatment has been devised.

CHAPTER II

THE LACRIMAL APPARATUS

ESSENTIALS OF THE ANATOMY

The lacrimal apparatus consists of the tear-secreting glands and their ducts, and the excretory conduction system which is composed of the puncta, the canaliculi, the lacrimal sac, and the nasolacrimal duct.

The lacrimal gland is incompletely divided into two lobes by the aponeurosis of the levator palpebræ superioris muscle. The orbital lobe lies in a fossa of the frontal bone, and the palpebral lobe lies in the loose tissues of the fornix. Both lobes are in intimate relationship with the eyeball. The ducts of the lobes lie in close contact or unite as common channels and open into the fornix. Operations dealing with one lobe may sever the ducts of both. The accessory lacrimal glands of Krause are distributed mainly in the superior fornix.

Tears are clear fluid consisting of about 99 per cent water, and 1 per cent salts and proteins. They are distributed over the conjunctiva and cornea by the winking movements of the lids. The lids therefore cleanse and moisten these surfaces. Mucus from the conjunctival glands mixes with the tears; when stringy it will collect at the inner canthus. Sebaceous secretions mingle with the tears; when excessive they collect at the inner canthus in yellow waxy masses. This is frequently observed in the eyes of poorly nourished children.

The plica semilunaris is a vertical fold of bulbar conjunctiva lying laterally to the caruncle. It may be demonstrated by turning the eye toward the temple. It and the caruncle direct the tears toward the puncta.

The lacrimal papilla is a small elevation on the posterior angle of the lid margin at the medial extremity of the line of lashes. It lies in contact with the eyeball normally. That of the upper lid is medial to that of the lower.

The punctum is in the summit of the papilla; it affords access to the drainage system. The vertical arm of the canaliculus lies in the midplane of the lid, and is directed at a right angle to the lid margin for about 1.5 to 2 millimeters. At the end of this arm is a dilatation or ampulla, from which the horizontal arm parallels the lid margin until it opens into the lacrimal sac, either independently or united with its fellow of the opposite lid. This opening is behind the medial palpebral ligament.

The lacrimal sac lies in the lacrimal fossa; it measures about 12 millimeters vertically, and about 6 millimeters horizontally. It is covered by skin, superficial fascia, muscle tissue and lacrimal fascia. The pars lacrimalis of the orbicularis oculi is attached to the posterior lacrimal crest, while the medial palpebral ligament is attached to the anterior. A reflected portion from the ligament is attached to the posterior crest.

The sac is continued downward through the nasolacrimal canal as the nasolacrimal duct, which terminates in an ostium in the inferior nasal meatus. Osseous irregularities and membranous folds are commonly found in these channels. The passages are lined with mucous membrane, and thus the conjunctiva is continuous with the nasal mucosa. Infections may extend from one structure to the other. Secretions from both the conjunctiva and the nose may lodge in the lacrimal sac, which is more prone to become diseased than any other part of the lacrimal apparatus.

The lacrimal gland is supplied by the lacrimal branch of the ophthalmic artery. The venous blood drains by way of the ophthalmic veins into the cavernous sinus. The lymphatic drainage is toward the preauricular gland. The nerve supply is derived from the trigeminus and sympathetic. The lacrimal nerve goes direct to the gland. The zygomatic branch of the maxillary anastomoses with the lacrimal nerve. Sympathetic fibers reach the gland by accompanying the lacrimal artery. Tears are secreted because of irritation due to drying of corneal epithelium, from irritation of the nasal mucosa, and in weeping.

The excretory passages are supplied by arteries mentioned in describing the vascular supply of this region of the lids. The venous blood drains into the veins of the face and into the pterygoid plexus. The lymphatic drainage is toward the submaxillary gland. The principal sensory nerve is the infratrochlear and its branches.

DISEASES OF THE LACRIMAL APPARATUS

The gland may undergo atrophy, hypertrophy or prolapse. A lobule or duct may form a cyst (dacryops). A fistula may come from it. Acute or chronic inflammation (dacryoadenitis) may attack it, and end in the formation of an abscess which can point toward the skin or the conjunctiva. Various neoplasms may invade its structure. Mikulicz's disease is characterized by bilateral chronic enlargement of the salivary, parotid and lacrimal glands. Each condition must be treated according to its nature and manifestations. Atrophy, hypertrophy or prolapse may call for extirpation of the gland; neoplasms must be removed.

The palpebral lobe may require extirpation. Local anesthesia is employed. The lid is fixed with an Ehrhardt clamp. An incision is made in the conjunctiva to expose the lobe, which is then dissected out (Fig. 61). Silk sutures are used to close the wound. White's ointment is applied and a pressure dressing is employed. The sutures are removed on the third day. In the dissection, the cutting of the ducts of the orbital lobe may be unavoidable. The orbital lobe is not to be removed without positive indications, because of the resulting complications of keratitis, conjunctivitis, or orbital hemorrhage.

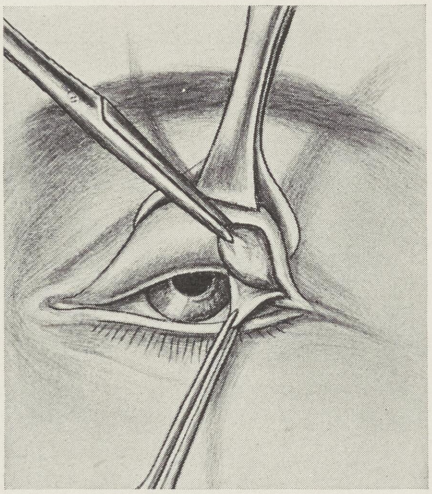


Fig. 61.—Incision in Conjunctiva. (After Wheeler.)

Pressure dressings may be tried for dacryops. For fistulæ, cauterization or dissection of the fistulous tract, or extirpation of the gland, either or both lobes, may be required. Dacryo-adenitis is treated with hot packs until the inflammation subsides. In the event pus forms and points, it may be evacuated on the conjunctival or skin surfaces, preferably the latter. For Mikulicz's disease de Schweinitz mentions arsenic; no specific is known. Major Wright, of India, reported two apparent cures by the use of x-rays.

DISEASES OF THE CONDUCTION APPARATUS

In diseases of the conduction apparatus, epiphora is usually the prominent sign. Where the punctum is not in contact with the globe no tears enter it. One of the operations for ectropion corrects the deformity. A galvanocautery puncture on the conjunctival surface with subsequent scar contraction may correct a slight eversion. To avoid wounding the canaliculus the puncture must be made 4 to 6 millimeters below the lid border.

The canaliculus may be occluded by a hair or a concretion. A hair is likely to protrude from the punctum, and can be removed with forceps. The canaliculus must be opened sufficiently to remove a concretion. Strictures form in the canaliculi, and require probing or slitting. A dilator is needed to open the punctum (Fig. 62).

The patient reclines on an operating table or chair. Cocain 4 per cent solution with adrenalin chlorid 1 : 10,000 is used for anesthesia. Several drops of this are instilled into the conjunctival sac near the inner canthus. The lid is everted by backward pressure with the thumb below the papilla. The point

of the punctum dilator is pressed and rotated into the punctum for 2 or 3 millimeters. A blunt needle is attached to a Luer syringe, and 2 or 3 drops of cocain solution injected into the canaliculus. The dilatation is resumed, and the handle of the instrument is lowered so that it lies horizontally along the lid border; it is then pressed and rotated into the canaliculus. A lacrimal probe is now substituted for the dilator (Figs. 63 and 64), and it is manipulated in the same manner.

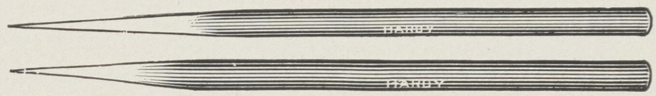


Fig. 62.

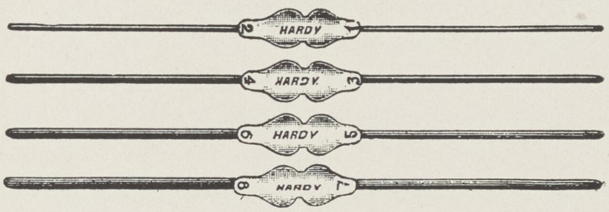


Fig. 63.

That the instrument is in the sac may be known in two ways: (1) Upon pressure toward the sac, the lid will follow the movement until the sac has been entered; (2) when the instrument enters the sac it comes into contact with the bony wall on the medial side, and the tactile sensation imparted to the examining fingers is that like tapping the face of a nickel coin with the point of a lead pencil. The upper punctum and canaliculus can be dilated similarly. Treatments are repeated two or three times a week until the epiphora is relieved.

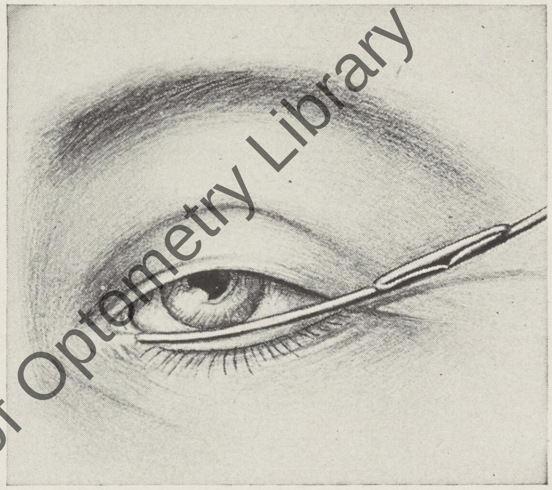


Fig. 64.—Probe in Canaliculus.

The nasolacrimal duct may be occluded. The probing is continued into the duct. After the instrument has entered the sac, the free end of it is rotated upward until the end in the sac is directed toward the floor of the nose on that side (Fig. 65). The instrument now rests against the medial extremity of the eyebrow. If more anesthetic is required it should be injected into the lacrimal sac. Unskillful manipulation might injure the membranous lining, create a false passage, or enter the orbit.

Having the probe in the position described, pressing it lightly against the brow and pushing it gently downward in the direction of the duct, it will be in

the channel if it stands unsupported; if it falls, it has not been in the duct. When it is properly directed, it may be pushed downward, occasionally rotating it about its axis, until it touches the floor of the nose (Fig. 66).

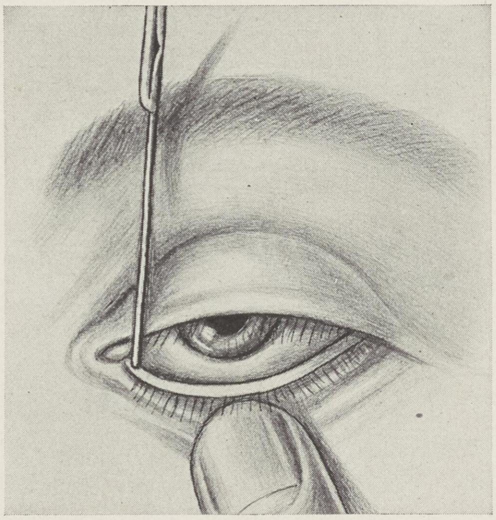


Fig. 65.—Probe in Lacrimal Sac.

Probing is usually begun with a No. 1 or No. 2 probe, and increasing sizes are used up to No. 4 or even No. 7, but seldom above No. 5. Occasionally a No. 3 or No. 4 probe will pass more readily than a smaller one. The passages are then syringed with boric acid solution, or 1:5,000 solution of bichlorid of mercury. The patient must not swallow these. The cocain and adrenalin solution may pass into the nose without previous probing. It is recognized by its bitter taste. In such instances the solutions may be used without probing. The treatments are repeated every three or four days until the epiphora improves, when the intervals between treatments are gradually lengthened.

Some operators advocate the rapid dilatation of the canaliculus and nasolacrimal duct with large-sized probes, and force a passage of large caliber at the first treatment. This is not the method for a novice.

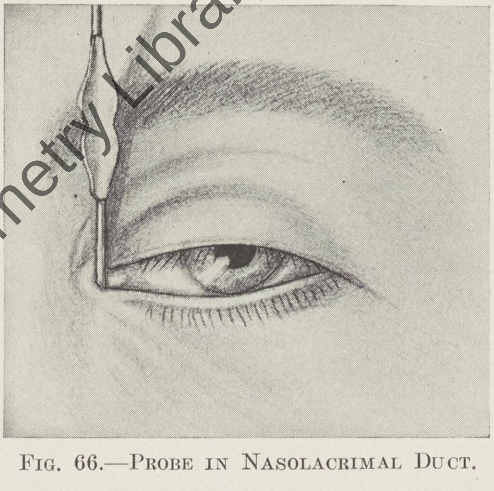


Fig. 66.—Probe in Nasolacrimal Duct.

Restoration of the patency of the passages is not always permanent; a recurrence of epiphora demands a renewal of the operative measures, or the institution of different treatment.

Because of occupation or distance from the physician, some patients are unable to obtain frequent treatments. Various plans have been devised to accommodate this class. Lacrimal cannulæ (Fig. 67) and styles are of similar shape; each is made of lead, silver or gold, and has two arms at right angles to each other: one of 2 to 2.5 centimeters length to fit into the nasolacrimal duct, and the other of 5 to 8 millimeters to rest in a groove made by slitting the canaliculus. They are designed

to be worn over considerable periods of time, but the epiphora generally recurs when their use is discontinued.

The canaliculus can be slit with a straight or curved Weber's canaliculus knife (Fig. 68). The punctum is dilated as for probing. The lid is fixed in position by the operator's thumb. The blade is inserted into the sac as is done in probing. The cutting edge is directed upward and backward, so that the *conjunctival angle* of the lid margin will be cut. The handle is elevated into a vertical position, as the probe was, and the blade cuts itself out of the canaliculus as it is elevated. The resulting groove will lie in the lacus lacrimalis (Fig. 69).

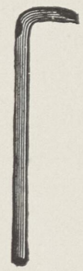


Fig. 67.

Where it is desired to pass the blade down the nasolacrimal duct, the cutting edge is first rotated backward, then outward, and finally forward, before it is passed into the duct. For this purpose Stilling's knife (Fig. 70) is preferable. The Weber blade may be passed into the canaliculus in several places where the caliber is contracted by strictures. In such cases it is unnecessary to cut out, but it is necessary to maintain the lumen by probing, as otherwise it will close during the healing process.

Fig. 68.

In some instances it is advisable to slit both canaliculi completely into the sac, so that the latter is opened when the lids are widely separated. This permits cleansing of the sac, but the epiphora will continue unless the nasolacrimal duct is patulous.

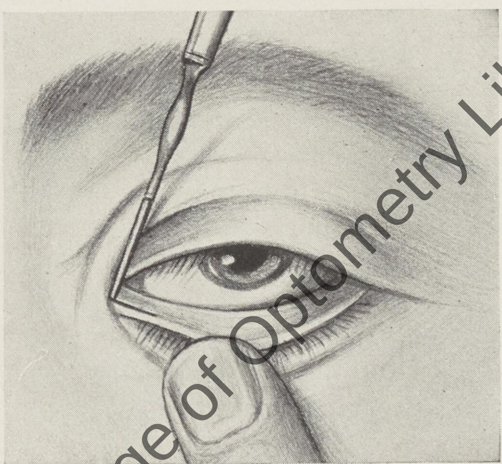


Fig. 69.—Slit in Angle of Lid Margin.

The excretory tear passages are developed embryologically from cords of cells. The cords do not always become canalized throughout. Sometimes the

ostium in the inferior nasal meatus is closed by a membrane. In the newborn this is manifested by a swelling over the location of one lacrimal sac; it is seldom bilateral. The surgeon must first be assured that the puncta exist; gentle pressure over the distended tear sac will cause a flow of fluid into the conjunctival sac. The lids are then drawn temporally, while the thumb of the opposite hand firmly presses the lacrimal sac against the bony fossa from above downward. This tends to rupture the membrane at the ostium of the nasolacrimal duct.

Should the plan fail, it is necessary to wait until the sac fills again before repeating the process. The nurse or parents can be instructed and directed to make this pressure several times a day until success or failure has been demonstrated. In the event of failure, it will be expedient to anesthetize the patient and pass a No. 1 probe. It is seldom necessary to repeat this. To guard against injury to the floor of the nasal fossa by the point of the probe, it is well to have two probes of equal length, one being laid alongside the nose with its point above the level of the nasal floor as a guide.

Fig. 70.

It has become an established practice to instill a solution of nitrate of silver into the eyes of the newborn, as a prophylactic against ophthalmia neonatorum. Many cases of tear sac occlusion date from birth, and these occlusions are prone to lead to infection and chronic dacryocystitis. Practitioners can render additional service when they habitually express the contents of the tear sacs through the nose after instilling the silver solution and at each of their postnatal visits.

Fig. 71.
NLD, Nasolacrimal Duct; *T*, Turbinate.

Epiphora does not always depend upon disease of the passages, but may occur from the presence of a foreign body on the cornea or conjunctiva, conjunctivitis, irritants in the atmosphere, and to some irritations of the nasal membranes. Such cases usually demonstrate, by the use of the handkerchief and by blowing the nose, that the excretory passages are open.

A few rare cases depend upon deformities of the lower turbinate bone. In acute rhinitis accompanied by great turgescence of the nasal mucosa, the membranes of the turbinate and the lateral nasal wall are swollen against each other (Fig. 71). The ostium is sealed and no secretion can escape. To probe a case of this kind is ineffectual, and more radical procedures, such as extirpation of the tear sac, cannot be defended. The mucosa of the meatus should be shrunken. After the acute rhinitis has subsided, adenoids and other obstructions to breathing should be removed. It may be necessary to break the turbinate bone away from the lateral nasal wall.

RELATED DISEASES

A **Mucocele** exists when the sac is distended by a viscid fluid that can be expressed into the conjunctival sac or into the nasal fossa. Atony is present when the mucous lining is atrophied or when elasticity is lost, and the sac is no longer emptied spontaneously.

Conjunctival secretions as well as tears are carried off by the excretory passages. As long as drainage is normal there is no colonization of incidental pathogenic organisms. While pneumococci are present in many healthy conjunctival and lacrimal sacs, they are innocuous during active drainage. Where secretions accumulate in the tear sac they become virulent.

Acute Dacryocystitis or lacrimal abscess is an acute pyogenic infection of a chronically diseased sac. The tissues surrounding the sac are inflamed, and constitutional symptoms of infection are present. Pain is often severe. The swollen, red and tender area extends from slightly above the medial palpebral ligament downward and outward to below the medial half of the lower lid. Suppuration progresses and fluctuation can be detected.

The abscess points at a little distance below the sac and ruptures spontaneously. Where this has not occurred the abscess should be incised. Cocain solution is instilled into the conjunctival sac. The incision is made with a narrow scalpel or Graefe knife from the lowest point of distinct fluctuation into the sac. The edge of the blade is directed forward, and the point of the knife is directed upward, inward, and slightly backward. The fistula or incision can be enlarged when necessary.

The abscess cavity should be irrigated by syringe with warm boric acid or 1:7,000 bichlorid of mercury solutions. It is advisable to dilate the canaliculi and again irrigate the sac through them. The sac is gently cleansed and dried with mops, and is then filled with White's ointment. A dressing is applied. At the next treatment the sac should be cleansed again, and an effort made to probe the nasolacrimal duct. This too should be irrigated with the solution. As the inflammation subsides, the fistula or incision usually heals. Probing ought to be continued until the secretions flow off naturally.

A diseased sac is a menace to an eyeball. The epithelium of the cornea and conjunctiva protect those structures. An abrasion affords the organisms an entrance beneath the protective epithelium, and an active process begins. Ulcus serpens is regarded with apprehension. If the organisms enter the globe, intra-ocular infection may be expected.

Fistula of the Lacrimal Sac occurs in neglected cases of lacrimal abscess. Where the conduction system is not obstructed, an effort should be made to close the fistula. Its course should first be explored by probing. Galvano-

cautery puncture deep into the fistulous tract may close it by scar formation. A narrow Graefe knife may be introduced into the passage and rotated to destroy the epithelial lining of the fistula.

Prelacrimal Abscess lies outside the sac, and mostly above the medial palpebral ligament. It can extend from a frontal sinus infection and be in communication with it. In this location (Fig. 72) it must be differentiated from other enlargements. The tears are drained naturally, and there is no pronounced epiphora. When the canaliculi are dilated, and fluid introduced into each finds its way into the nose or throat, attention is directed to an abscess outside the sac. Any communication with the sac is by way of a fistulous tract.

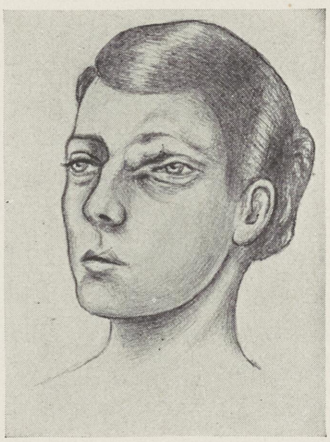


Fig. 72.—Prelacrimal Abscess.

An inflammatory swelling, mostly above the ligament, without epiphora, is reasonably diagnostic. The tissues about the tumor are infiltrated with novocain solution. The abscess, not the sac, is incised, its cavity is lightly curetted and thoroughly cleansed, White's ointment is applied, and the wound is dressed without sutures. Daily attention is advisable until healing is completed.

OPERATIONS ON THE TEAR SAC

Dacryocystectomy, or extirpation of the lacrimal sac, is indicated for an incurably diseased sac, and to guard against infection when it is proposed to open the eyeball. The instruments required are a small sharp scalpel, two fixation forceps without locks (see Fig. 167), small curved scissors, small curets (see Figs. 12 and 13), strabismus hooks (see Fig. 246), blunt dissector, punctum dilator (see Fig. 62), No. 3 lacrimal probe (see Fig. 63), Müller's or Wilder's lacrimal sac retractor, fine hemostatic forceps, and small curved needles.

Anesthesia may be local or general; the latter is preferred. If local, freshly prepared solution of novocain and adrenalin is infiltrated deeply into the area about the sac. The field is prepared for surgical asepsis, and the operator wears rubber gloves. Sterile petrolatum is applied to the conjunctival sac, and the lids are sealed with a vertical strip of adhesive tape, or by temporarily suturing them together by their skin surfaces.

The surgeon stands at the right side of the patient's head. The left thumb immobilizes the tissues in their natural position above the medial canthal liga-

ment by firmly pressing them against the underlying bone. The anterior lacrimal crest is located.

Beginning 3 millimeters above the upper border of the ligament, an incision is made alongside the crest to the upper opening of the nasolacrimal bony canal (Fig. 73). The skin is dissected laterally from the incision, and the superficial fascia, muscle and lacrimal fascia are each divided with scissors and dissected back separately. The dissection can be made with the points of the closed scissors or the blunt dissector.

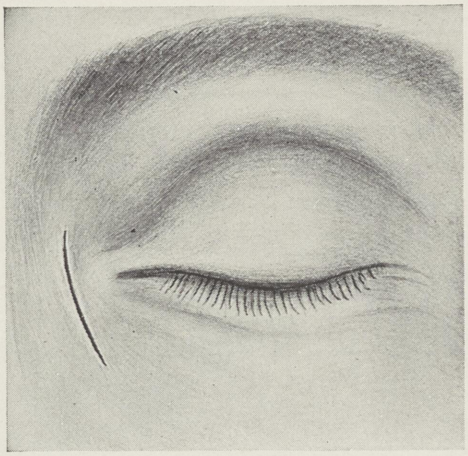


FIG. 73.—DACRYOCYSTECTOMY.

The lacrimal fascia must not be mistaken for the sac wall. When the latter has been exposed it will present into the wound as a rounded bluish body, especially after the ligament has been severed (Fig. 74). The lacrimal retractor may now be used, or an assistant can keep the wound open with fixation forceps and strabismus hooks. Dissecting closely against the lateral wall of the sac, the canaliculi are exposed. When there is any difficulty in locating these, the puncta can be dilated and probes passed into the sac.

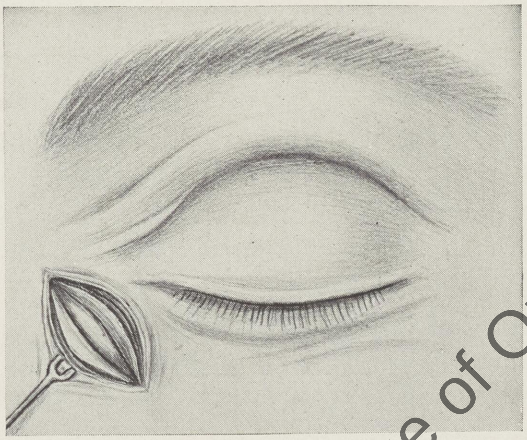


FIG. 74.

The canaliculi are cut as far from the sac as can be done without retracting the puncta. The dissection is continued until the lateral wall is free. Next the fundus is dissected out, and then the medial wall. By seizing the fundus with fixation forceps and lifting it up, the posterior wall is readily detached; the sac will then lie outside the wound.

The original incision is extended downward and outward to permit a working space into the nasolacrimal bony canal. The duct is divided with curved scissors as deeply within the canal as possible. The canal is next curetted with small sharp and serrated chalazion curets. The canal is *dried* with gauze or cotton mops.

A probe is passed into the amputated sac, which is then inspected for incom-

plete extirpation. Remnants are removed by curetting, or the infection will continue. The bony fossa is packed or mopped until oozing has ceased. A drain is not needed except in cases where surrounding tissues are diseased, or when the oozing cannot be completely checked. If used, it should be removed as soon as conditions permit.

The wound is closed with a continuous buttonhole suture, the needle in each instance being passed at a higher level on the nasal side than on the temporal, to prevent sagging from the weight of the lid (Fig. 75). Dermol or silk may be used. The eyeball ought now to be inspected; if undamaged, apply White's ointment to the conjunctival sac and to the line of the incision. A short roll of gauze is applied to press the soft tissues into the vacated fossa, and a pressure dressing of cotton-wool pads is used. The sutures are removed in four or five days.

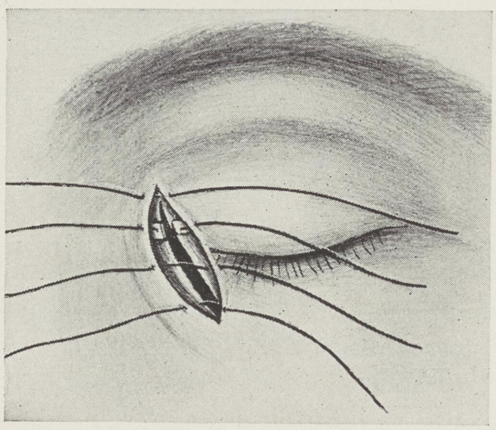


Fig. 75.
Note direction of sutures. (After Wheeler.)

Where the puncta are diseased, they and the remainder of the canaliculi can be destroyed with the galvanocautery. While this operation eradicates the diseased tissue, it does not abolish the epiphora. The majority of patients cease to complain of it. The lacrimal gland can be removed where epiphora persists.

Dacryocystorhinostomy is preferred by some surgeons because it provides drainage for the tears; it also opens an avenue for infection from the nasal cavity. The incision is made along the anterior lacrimal crest as in the former operation. The dissection is made along the medical wall of the sac only, with the intent of detaching the periosteum from the bone on that side. A probe is passed into the sac as a guide, and the bony nasal wall is resected. A flap is formed of the nasal mucous membrane, and a window is made in the wall of the sac opposite the location of the nasal membranous flap.

By this method the tears are "short-circuited" into the nose. This operation cannot cure a disease of the nasolacrimal duct, which usually coexists. It does not protect the eyeball in operations that open it. However, the canaliculi can be ligated, and the puncta can be temporarily sealed with the galvanocautery previous to operations on the globe.

CHAPTER III

THE ORBIT

ESSENTIALS OF THE ANATOMY

The bony orbital walls are formed from the frontal, zygomatic, maxillary, lacrimal, ethmoidal, palatal, and sphenoidal bones. The orbital cavity has a roof, side walls and a floor. The shape of the orbit is molded by the growing contents in prenatal life.

The roof of the orbit is the floor of the frontal sinus; its floor is the roof of the maxillary sinus; and the medial orbital wall is the lateral wall of the ethmoidal and sphenoidal air-cells. The frontal and maxillary sinuses and the anterior ethmoidal cells are in relation to the anterior two-thirds of the orbit, while the posterior ethmoidal and sphenoidal cells are in relation to the posterior third.

The anterior opening is contracted, so that the widest part is about 1 centimeter back of the anterior face of the orbit. The crest is the narrowest circumference of the opening. Important vessels and nerves appear upon the face from the orbit by way of foramina and incisure. The supra-orbital nerve and artery and the frontal vein pass through the supra-orbital foramen or notch. The supratrochlear nerve appears medial to the supra-orbital. The frontal vein receives, in or near the notch, the frontal diploic vein which drains the membrane of the frontal sinus. The infra-orbital nerve and artery appear on the surface through the infra-orbital foramen. The possibilities for extensions of infections from the surface to the orbit are many.

Two or three foramina are located in the suture uniting the frontal and ethmoidal bones. The anterior is the most constant and transmits the anterior ethmoidal nerve and artery. The posterior transmits a nutrient artery to the membrane of the ethmoid cells, and sometimes a small nerve. The third is seldom found, but when present lies between the other two, and transmits an artery. These nerves are branches of the nasociliary from the ophthalmic division of the trigeminus. The arteries are branches of the ophthalmic. Lymphatic vessels are probably present.

The posterior third of the bony orbit has three major openings, namely, the optic canal, and the superior and inferior orbital fissures. The optic canal at the apex of the orbit transmits the optic nerve and the ophthalmic artery

with the sympathetic plexus upon it. The superior orbital fissure lies between the wings of the sphenoidal bone. All of the nerves that enter the orbit except the optic and the infra-orbital, the ophthalmic vein and sometimes the anterior meningeal artery pass through this fissure.

The inferior orbital fissure lies between the greater wing of the sphenoidal and the palatal bones. The maxillary nerve and the infra-orbital artery pass through it to enter a groove on the floor of the orbit. About halfway forward this groove acquires a roof, and becomes a canal which terminates at the infra-orbital foramen. The inferior fissure also transmits communications between the inferior ophthalmic vein and the pterygoid plexus. After head injuries, blood may enter the orbit through the fissure and work its way forward to undermine the conjunctiva.

The periorbita is the periosteum of the orbit; it is continuous from the dura mater through the optic canal. The membrane is loosely attached to the bone, and for that reason is easily separated from it by effusions, exudates and extravasations which may arise in neighboring structures.

The contents of the orbit comprise the eyeball, the optic nerve, the lacrimal gland, the ciliary ganglion, nerves, blood-vessels, muscles, fascia and fat. Fat lobules fill the space not occupied by essential structures. The fascia is connective tissue that possesses somewhat elastic properties. It constitutes a framework for maintaining the principal contents in position. The fascia is attached to the crest of the orbital margin anteriorly as the orbital septum. It supports the orbital fat.

The fascia bulbi, or Tenon's capsule, is an important subdivision of the fascia. It surrounds the globe and acts as a barrier against the migration of infections from the orbit to the eyeball, and from the eyeball to the orbit. Because of the peculiar interlacing of the elastic fibers, the globe moves in its bed of fat without friction. The capsule invaginates the extra-ocular muscles as they are inserted into the globe, and it is perforated by the optic and ciliary nerves, the ciliary arteries and the venæ vorticosæ. The fascia is loosely united to the surface of the globe by fine fibers which constitute the episcleral tissue.

The Examination of the Orbit.—External injuries, edemas of the lids and localized swellings are evident to inspection. The position of the eyeball and its motility are easily determined if but one orbit is affected. Enophthalmos, proptosis, and the direction of gaze of such an eye are to be estimated. The movements of one eye are compared to those of the other.

Where an eyeball protrudes, digital pressure will determine if it can be replaced, and if such pressure produces pain. The space between the orbital margin and the globe should be palpated for tumors, cysts, fluctuation and tenderness. The size and mobility of the two pupils are compared. Differences

in congestion and chemosis of the conjunctivæ of the two eyes will be observed, especially in the circumcorneal region. Disturbances in vision require ophthalmoscopic and field study.

The accessory air cavities of the nose are lined with mucoperiosteum continuous with the nasal mucosa. Channels from the sinuses open into the several meatus. Congestion of the membrane of the turbinates may occlude the ostia of the channels, and so arrest drainage from and aëration of the cavities. Stagnant secretions and vitiated air irritate the sensory nerves supplying the tissues. This is capable of producing headaches, and the pain may be referred to the eyeball.

A rapidly established eyeball pain with brow ache which appears without antecedent injury or disease suggests occlusion of the ostium of a sinus. There may or may not be tenderness over any sinus. When the nasal membranes are congested, or contact points are found, a few drops of 1 per cent cocain solution are sprayed or swabbed on that surface. If the eye pain and headache are relieved within a reasonable time, the nose rather than the eye should be treated.

DISEASES OF THE ORBIT

Orbital Periostitis is due to injuries, syphilis, more especially in adults, tuberculosis, more especially in children, and to migrating purulent infections. The *signs* are deformity of the orbital margin with redness of the overlying skin. An immovable hard swelling can be palpated. The eyeball may be displaced forward or to one side and be limited in motion. The lids and conjunctiva are often edematous. A fistula may be present. Subjectively, there are tenderness to pressure and deep-seated pain. Constitutional symptoms are usually present. The disease pursues an acute or a chronic course. Gummatous thickening of the periosteum may occur.

Symptoms vary according to the location of the affection in the orbit. Where the process is deeply situated the displacement of the globe is more pronounced, and the symptoms are more intense. The pain is more neuralgic in character, and will probably be aggravated at night. When the periosteum is detached from the bone by suppuration, caries or necrosis takes place. Usually the temporal or the inferotemporal margin of the orbit is affected. The abscess extends to the surface and ruptures. Because of the destruction of bone, healing is accompanied by deformity.

Treatment of periostitis is directed toward the cause, relief of the local condition, and building up resistance. Where the disease is due to sinusitis, the infected cavity must be drained. Hot moist compresses are indicated; in the earliest stages the process may be aborted, while in later stages the develop-

ment is hastened. Because of proptosis or inability to close the lids, the cornea must be protected by sterile petrolatum and dressings, or by bandage.

When the abscess is located it must be drained. An incision is made at the superior medial angle of the orbit or at the inferior lateral angle. The incision should go through the periosteum, which is then elevated from the bone as far as the abscess. The pus is evacuated, carious and necrotic tissues are removed, and a drain is inserted.

Orbital Cellulitis or retrobulbar abscess is due to injuries with infection, extension of infections from neighboring cavities, the face or eyelids, septic emboli, thrombophlebitis, dental caries or infections, suppurative meningitis, the exanthemata, or almost any septic condition.

An acute cellulitis is an active infiltrative process with constitutional reactions. The lids are edematous and the conjunctiva is chemotic. The globe is displaced, and attempts to move it are ineffectual and painful. The patient has diplopia. The optic nerve is inflamed, and the ophthalmoscope will reveal optic neuritis. An examination of the visual fields discloses an enlarged blind-spot and a central scotoma for red and green. Vision is reduced early, and it may be lost.

When the process continues to suppuration the abscess will point and rupture. The symptoms subside rapidly. Loss of vision occurs because of optic nerve involvement, or the contents of the eyeball may undergo suppuration. The preauricular gland is usually swollen. The infection may enter the cranial cavity and terminate in the death of the patient from meningitis or cerebral abscess.

Disease of the accessory cavities of the nose is often a source of orbital cellulitis. The extension is facilitated by a dehiscence in a partition wall. Sometimes ethmoiditis produces a serous infiltration of the orbital tissues without evidence of inflammation, but with signs of intra-orbital pressure. The eyeball is proptosed, and attempts to move it are only partially successful, but without much pain. The pupil is dilated. An examination of the sinuses should be made. A negative roentgenogram does not exclude sinus disease. Transillumination offers suggestive, and occasionally positive evidence. Pus in the anterior or posterior nares is more dependable.

Diagnosis of orbital cellulitis is made from a history of infection, lid edema, proptosis, limited ocular rotations, pain to backward pressure on the globe, unilaterality, early impairment of vision, and abscess formation. It must be distinguished from cavernous sinus thrombosis, pulsating exophthalmos, serous tenonitis, panophthalmitis, tumors of the orbit, orbital emphysema, and paralysis of extra-ocular muscles.

Treatment.—Wounds of the orbit require antiseptic cleansing and drainage.

Foreign bodies must be removed. Diseases of the sinuses should have energetic treatment. Hot fomentations over the orbit and leeches (Fig. 76) to the temple are indicated. As soon as pus can be detected it must be evacuated. Pus may burrow forward and point along the orbital margin. In deeper situations it will press against the eyeball; the position and deviation of the latter will suggest the location of the pus. Where the abscess is well forward, a simple incision inside the orbital margin and parallel with it will reach and release the pus. The incision must be ample for drainage, and for the insertion of a drain.

Should the abscess be more deeply situated, an incision can be made as before; the soft tissues can then be divided toward the pus by blunt dissection with a narrow-bladed artery forceps. Care must be exercised to avoid injury to the contents of the orbit, especially to the trochlea. A purulent ethmoiditis may communicate with an orbital cellulitis through a dehiscence in the bone. A fenestrated drainage tube is carried from the orbit through the bone and out at the nose. With this arrangement irrigation is readily accomplished, and it should be continued until healing is established. Ample drainage tends to prevent the extension of the process to neighboring structures, such as the cavernous sinus. Symptomatic and supportive treatment must be given as indicated.

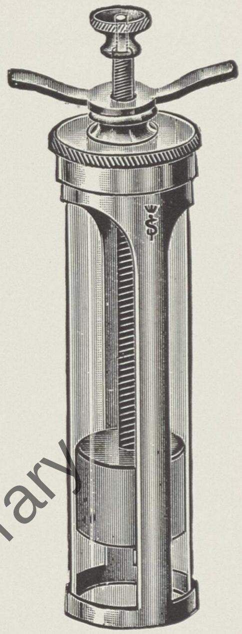


Fig. 76.—Artificial Leech.

Special caution is enjoined concerning any circumscribed swelling at the orbital margin above the medial canthal ligament. It may occur in periostitis or cellulitis, which are inflammatory. A mucocele from the frontal sinus or from an anterior ethmoidal cell will have a similar appearance. An encephalocele pulsates as brain tissue does, and must never be molested. A meningocele is filled with cerebrospinal fluid and may pulsate. It is covered by the pia-arachnoid but not by dura, and can be reduced by pressure, but probably with cerebral symptoms such as nausea and dizziness; it possesses fluctuation and is translucent to transillumination. Mucoceles, encephaloceles and meningoceles are immovable and noninflammatory (see Fig. 72).

A dermoid cyst appears here or at the superior lateral angle of the orbit. It is formed from an inclusion of epiblast between growing bones, and therefore is located at a suture line. It is movable and is not tender. Dermoid cysts are removed under general anesthesia, since they extend deeply into the orbit. An incision is made through the shaved eyebrow concentric with the orbital margin and as deep as the periosteum. The tumor should be dissected

out without rupture of its walls, for where any dermoid tissue is left a recurrence may be expected. The wound is closed and a pressure bandage is applied until healing is established.

In the aged a hernia of fat frequently appears from beneath the superior medial angle of the orbit.

A prelacrimal abscess is inflammatory, and is often related to sinus disease. Hematomata, osteomata and other tumors may appear in this region. When satisfactory palpation of the orbital margin cannot be made through the lids, the conjunctiva can be cocainized, and then palpation can be made from inside the lids.

Vascular tumors, including aneurysms and angiomata, occur in the orbit and produce a variable proptosis. The globe can be pressed back, but comes forward when stooping or straining. Whenever the eyeball is in danger, these masses should be removed. Osteomata and exostoses cause deformity, and produce diplopia when they crowd the globe. They are removed when indicated. When they are due to syphilis, antisyphilitic treatment should remove them. Gummata of the orbit require the same treatment.

Fig. 77.—Proptosis; Krönlein Incision.

A wide variety of new growths have been found in the orbit. Tumors of the intra-orbital portion of the optic nerve usually produce proptosis (Fig. 77). These neoplasms, even when situated behind the globe, have been removed with preservation of the eyeball for the cosmetic effect. The operations of Krönlein and of Knapp have been designed for that purpose. The former is accomplished by a resection of the lateral wall of the orbit, and the latter by a temporary detachment of the medial rectus muscle.

The anterior face of the temporal margin of the orbit is often so deeply concave that the eyeball is visible behind its equator when the cornea is rotated nasally. Much of the retrobulbar space is available as an operative field. An incision can be made behind the lids after they are separated by a canthotomy.

Malignant Tumors arising in the orbit or invading it from a sinus require exenteration of the orbit. The cautery should be used freely. All suspicious tissue must be burned away.

Emphysema of the orbit is characterized by diffuse puffiness of the lids and by crepitation. Inflammation is absent, but ecchymoses may be present. The

eyeball is pressed forward, but the degree of proptosis is deceptive because the swollen lids contract the palpebral aperture. The air is beneath the skin of the lids as well as in the cellular tissues. A fracture of some bone provides a portal of entry. The air can only enter the orbit under pressure, as from blowing the nose. The globe can be pressed back without pain, but will come forward again with inflation of the orbit. The patient is to be cautioned against blowing the nose, sneezing or straining. A pressure bandage is indicated.

Hemorrhage may occur into the orbit. Fractures of the skull, especially of the base, are prone to produce it. The blood extravasates forward and appears as an ecchymosis of the lids or as a subconjunctival hemorrhage. In basal fractures, blindness may ensue from injury to the optic nerve in its canal. The direct pupillary light reflex is lost, but the indirect is preserved, along with that to convergence. The optic disk atrophies. The absorption of light hemorrhage is promoted by a pressure bandage, and potassium iodid is given. To drain the blood away, the orbit is opened surgically as suggested for the treatment of orbital cellulitis.

Fig. 78-A.—Left Cervical Sympathetic Paralysis. (After Fuchs.)

In external ophthalmoplegia the globe is proptosed and limited in motility. It can be pressed back, but almost immediately "sinks" forward again.

Enophthalmos is a backward displacement of the eyeball, with narrowing of the palpebral aperture. After section or paralysis of the cervical sympathetic nerve, or after extirpation of the superior cervical ganglion, Horner's syndrome or sympathetic ophthalmoplegia, consisting of enophthalmos, miosis and pseudoptosis, is present (Fig. 78-A). In lethargic encephalitis this syndrome may be present; one or both eyes may be affected. Paralysis of accommodation is occasionally associated.

Traumatic enophthalmos has a history of injury. The lids sink backward and the palpebral aperture is narrow. True ptosis may or may not exist. Ocular rotations may be limited or not, and diplopia may be present. The signs depend upon the degree of displacement. The globe may be far back in the orbit or in the antrum.

Enophthalmos does not always immediately follow injury. At first there may be exophthalmos, from extravasation of blood or infiltration of serum. As resolution progresses the tissues contract and the globe recedes. In other cases the eyeball is immediately displaced by direct force. The treatment is symptomatic.

Tenonitis is usually a serous inflammation of the fascia bulbi which a characterized by transient recurring fluctuating edema of the bulbar conjunc-

tiva, fixation of the globe in the primary position, and some degree of proptosis. The eyes are affected alternately, and perhaps several times each, although both may be affected simultaneously in late stages. The lower half of the edematous bulbar conjunctiva may conceal the lower lid. The exposed membrane is dry, dusky, smooth and tense.

The disease may occur in patients who are subject to recurring attacks of acute arthritis; attempts to actively or passively move the eye are attended by severe pain. Vision is unaffected in uncomplicated cases. The fundi are usually normal; where the inflammation extends far back and implicates the nerve sheaths, the retinal veins are engorged, the arteries are contracted, and optic neuritis or even papilledema may be present.

Serous tenonitis results from exposure, influenza, the exanthemata, focal infections, orbital disease or endogenous infections. An exudation occurs into Tenon's space. This fluid is albuminous and fibrinous. There is no suppuration. Corneal ulceration and iritis are apt to complicate cases of prolonged duration. A chronic plastic form accompanies panophthalmitis and follows some cases of iridocyclitis; permanent adhesions are formed.

Diagnosis of tenonitis is made on fixation of the eyeball in the primary position, alternating proptosis, and extruding edema of the bulbar conjunctiva. Orbital abscess and orbital cellulitis are acute unilateral conditions with proptosis and more or less fixation of the eyeball. Cavernous sinus thrombosis begins on one side and extends to the other. Episcleritis and scleritis do not immobilize the globe. Brawny scleritis is unilateral. Pseudotumor and tumor of the orbit are unilateral.

Treatment of serous tenonitis avoids the attempt to secure drainage by incision or puncture. Moist hot compresses and pilocarpin sweats are indicated. Sodium salicylate in doses as large as the patient can tolerate are given by rectum or by intravenous injection. Potassium iodid should be given as soon as the active symptoms begin to subside.

Corneal complications must be prevented if possible. An ointment of dionin 5 per cent in sterile petrolatum is suggested. The prognosis should be guarded.

Pseudotumor of Orbit.—The globe is displaced forward and usually to one side; it is restricted in mobility or fixed in position. It is unilateral. The onset may be rapid or slow and insidious. The symptoms vary in intensity, and there are few or no remissions. Both lids swell. The conjunctiva is chemotic and may protrude between the lids. The eyeball is inflamed. Vision is unaffected, but diplopia may be present. The duration is from a few weeks to several months. The signs disappear spontaneously or after medication with mercury, potassium iodid or salvarsan. At operation no tumor is found. The

diagnosis is difficult. The treatment is symptomatic or antisyphilitic. The cornea must be protected.

EXTRA-ORBITAL DISEASES

There are several diseases which are not classified among those of the eye, but because of ocular or orbital effects are discussed in textbooks on ophthalmology. They emphasize the anatomical environment.

Cavernous Sinus Thrombosis arises from an extension of phlebitis in other blood sinuses, from infections, and in debilitated subjects. The cavernous sinus receives blood from the face, forehead and eyelids, from the nose and accessory cavities, from the palate, gums, tonsils and pharynx, from the mastoid region and inner ear, and by way of the blood sinuses from intracranial structures. Pathogenic infection in any of these situations causes this disease.

Symptoms and signs develop rapidly as a rule. A chill may be the first indication. The fever usually exhibits a septic curve. Drowsiness, nausea and mental derangement may be present. Locally, there are progressive swelling of the lids, chemosis of the conjunctiva, proptosis, immobility of the globe, and edema of the mastoid region. When these signs appear on the side of the head opposite to a localized infective lesion, or are greater on that opposite side, attention is directed to the cavernous sinus rather than to the orbit. Some or all of the eye-moving muscles, especially the lateral rectus, are usually paralyzed. The fundus changes consist of greatly distended and tortuous retinal veins, hemorrhages, edema of the retina and optic neuritis.

The opposite cavernous sinus will become involved in from two to four days in about half of the cases. Meningitis is apt to occur. The disease is of short duration and ordinarily terminates in death. In recent years surgical treatment has been attended with encouraging results. The medical treatment is palliative and symptomatic.

Pulsating Exophthalmos is generally due to trauma, and in most instances is accounted for by a rupture of the internal carotid artery in the cavernous sinus. As arterial blood is forced into the sinus a pulsation is imparted to the contents of the orbit. Blood is propelled backward through the ophthalmic veins and their tributaries. Enlarged veins appear upon the surfaces of the swollen lids and surrounding areas, and the veins of the conjunctiva and retina are engorged. Hemorrhages may occur in the retina. In many cases there is an optic neuritis that leads to diminished vision, or to blindness if the condition is not relieved.

The globe is proptosed, but can be pressed backward in the orbit; it is proptosed again when the pressure is relaxed. There is limitation of movement of

the globe. Pulsation is manifest to palpation, and a murmur or bruit is heard over the globe, the temple, and sometimes over the entire skull. It remits during cardiac diastole. The patient may describe this murmur as similar to intermittently escaping steam. Hearing may be impaired. Orbital pain may be present.

A *diagnostic feature* is that the symptoms can be ameliorated or abolished by pressure on the carotid of the same side.

Treatment is often designed accordingly. The patient is put to bed. Digital pressure is applied at intervals over the carotid for a period of time, enough to suppress the bruit, but not necessarily enough to occlude the artery. If the plan promises success, ligation of the artery is indicated. Ligations have also been done on the superior or inferior ophthalmic veins or on the angular vein. Iodids are given.

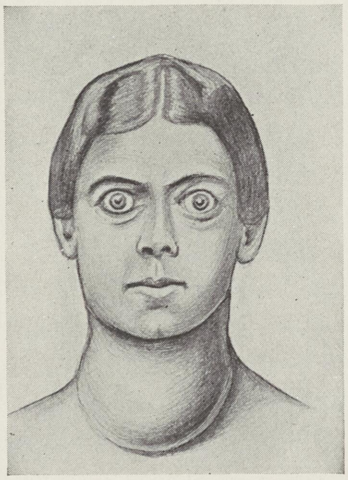


Fig. 78-B.—Exophthalmic Goiter; Dalrymple's Sign.

Intermittent Exophthalmos somewhat resembles the pulsating form; it is exaggerated by pressure on the jugular vein of the same side. It has been found to be due to varicose veins in the orbit, and ligation of them may be indicated or not according to the severity of the symptom.

Exophthalmic Goiter or Basedow's disease is characterized by excitation of the sympathetic nervous system. The involuntary palpebral muscles are stimulated, and the lids separate in an expression of astonishment (Dalrymple's sign, Fig. 78-B); after looking upward, the upper lid does not keep pace with the cornea on looking downward, and the sclera shows above the cornea (von Graefe's sign); the frequency of winking is diminished (Stellwag's sign); and there is a weakened power to converge (Möbius' sign). Exophthalmos may be actual, or apparent from the widely separated lids. The tense state of the upper lid may be judged by the difficulty experienced when attempting to evert it (Gifford's sign). The thyroid gland is enlarged. Tachycardia is present.

Because of the infrequent winking and imperfect closure of the lids, the cornea is liable to desiccation and consequent ulceration. The eyes should be bandaged at night when there is any sign of this complication; if this does not protect, a partial tarsorrhaphy should be performed. Paralysis of eye-moving muscles is functional, deviations are inconstant, and no operation is indicated. Vision is unaffected unless the cornea is damaged.

Acromegaly is often accompanied by proptosis to a degree that the globe is

readily dislocated outside the lids. The same protection for the cornea is indicated, when needed, as in Basedow's disease.

Exophthalmos has been noted in cases of renal retinitis, pituitary disease, oxycephaly, and rarely in other diseases.

ENUCLEATION OF THE GLOBE

The indications for removal of the eyeball are divided into three classes: That in which the removal is imperative, border-line cases, and that in which enucleation is elective.

1. The first class embraces malignant intra-ocular tumors, impending sympathetic ophthalmitis, eyeballs that have been extensively lacerated or otherwise injured beyond repair, and tuberculosis of the choroid when it persistently advances regardless of medical treatment. The possible exception exists when one eye is blind and the other harbors a malignant growth, and metastasis is suspected or already evident; the patient has not long to live.

2. Border-line cases embrace glaucomatous eyes that are blind and remain painful regardless of all treatment, chronic inflammation of the uveal tract associated with blindness and pain, acute iridocyclitis, shrunken globes that are subject to secondary inflammatory reactions, eyes filled with blood, and those that retain an irritating foreign body.

3. Operative removal for cosmetic reasons includes quiet shrunken globes, staphylomata of the sclera and cornea, and blind disfiguring eyes.

Enucleation is *contra-indicated* in panophthalmitis except in the earliest stages. After the disease has developed, the globe can be drained through the opened cornea, or its contents should be cleanly eviscerated.

The *instruments* needed for enucleation are: speculum (see Fig. 166) or retractors (see Fig. 204), fixation forceps (see Fig. 167), squint hook (see Fig. 246), small curved scissors, enucleation scissors and needle holder (see Fig. 178).

General *anesthesia* is preferred. Where ether is contra-indicated, nitrous oxid gas may be given. Local anesthesia may be used by election. A few drops of cocain and adrenalin solution are instilled into the conjunctival sac. Novocain 2 per cent solution in adrenalin 1:10,000 is injected subconjunctivally, into the insertions of the recti muscles, and as near the posterior aspect of the globe as possible in order to anesthetize the ciliary nerves. The ciliary ganglion can be anesthetized. The novocain will take effect in from twenty to thirty minutes.

The conjunctival sac is irrigated with 1:8,000 bichlorid of mercury solution. The lids are separated with speculum or retractors. The conjunctiva is

picked up and opened near the cornea (Fig. 79); it is divided with small curved scissors as near the cornea as possible.

A suture is passed through the conjunctiva at the medial side to retract that membrane while it and the episclera are being separated from the sclera. It is important that the closed blades of the scissors shall be inserted beneath the conjunctiva and not into the canthus when the undermining is attempted.

As soon as the recti muscles are exposed, they are raised in turn from the sclera with the squint hook or forceps and their tendons are divided with scissors (Fig. 80). The fascia bulbi is then undermined back to the optic nerve. This is easily done except where the membrane has adhered to the sclera because of old inflammation or subconjunctival injections of medicinal solutions; in such cases the dissection may be found difficult.

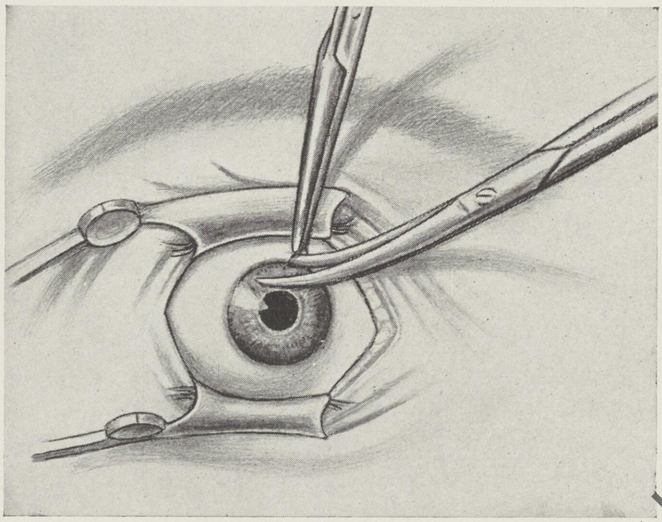


Fig. 79.—Enucleation of Globe. (After Meller.)

After the globe has been freed from conjunctiva and the fascia bulbi, the retractors are pressed backward into the orbit; a fascia-free globe will promptly prolapse outside the lids. The next step is to sever the optic nerve. This is done ordinarily as close to the eyeball as possible; in cases of intra-ocular tumors, since early extra-ocular extensions may occur along the optic nerve, it should be cut off as far back in the orbit as practicable.

The stump of the tendon of the medial rectus is seized with fixation forceps and the cornea is rotated temporally. The closed blades of the enucleation scissors are introduced behind the globe. By feeling around, the stretched nerve can be located. The blades are then opened, and the nerve is located between them; it is severed with a single cut (Fig. 81). Where the globe has collapsed from injury or disease, the locating of the nerve may be difficult, and the posterior segment of the globe may be amputated instead. This is not fatal to successful healing, since it is done so deliberately by some surgeons. But all uveal tissue must be removed.

Having disposed of the nerve, the surgeon draws the eyeball forward and toward the nose, and divides the tendons of the oblique muscles, while the

assistant presses a gauze sponge into the cavity as soon as the globe has been rotated nasalward.

The hemorrhage is arrested with packs. A purse-string suture is passed through conjunctiva at the free end of each rectus muscle in turn, and tied; the free ends of the suture are not cut too short. White's ointment is placed in the conjunctival sac, and a pressure dressing is applied. The wound is dressed daily. The suture is removed under cocain in five or six days. The patient is instructed to cleanse the conjunctival sac daily.

A prosthesis should be fitted and worn as soon as healing permits, usually in from two to four weeks. Undue delay invites deformity of the lids and shrinking of the conjunctival culdesac. With these complications it is difficult to adjust a glass eye for comfortable wear.

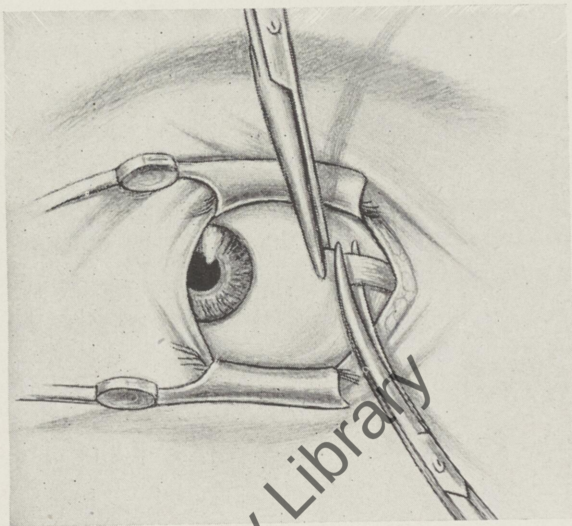


FIG. 80.
(After Meller.)

Various substances such as gold, glass and ivory spheres, fat, cartilage, decalcified ox bone and so on have been contrived and recommended to replace the globe in the socket. These tend to work out eventually, and often prove irritating.

A glass eye moves to some extent, not by a pull of the muscles, but rather by a push. To observe this, remove the prosthesis and watch the vacated space while the patient rotates the remaining eye in various directions.

Substitute Operations.—*Abscission of the Cornea.*—The cornea can be incised crucially when it is necessary to have simple drainage from an active intra-ocular abscess. The dressings should be loosely applied, so as not to interfere with free drainage of the pus.

When the general symptoms and ocular signs of inflammation are not too violent, the cornea may be abscissed. The conjunctiva is undermined surrounding the cornea, as it is for enucleation. A purse-string suture is run through the conjunctival flap. A horizontal elliptical section is cut from the cornea, the lens is delivered, and the suture is drawn tightly and tied. Dressings are applied with moderate pressure.

This operation is frequently performed for corneal staphyloma. It gives the patient an excellent stump for a prosthesis, but there is always danger of

sympathetic irritation or even of actual inflammation. It is generally safer to eviscerate.

Evisceration.—This operation is the one of choice for cases of panophthalmitis. General anesthesia is preferable to local on account of the congested state of the orbital tissues and the presence of a plastic tenonitis. The operation is begun as for enucleation. The conjunctiva is incised around the cornea, and is dissected from the sclera for a distance of 6 to 8 millimeters from the limbus.

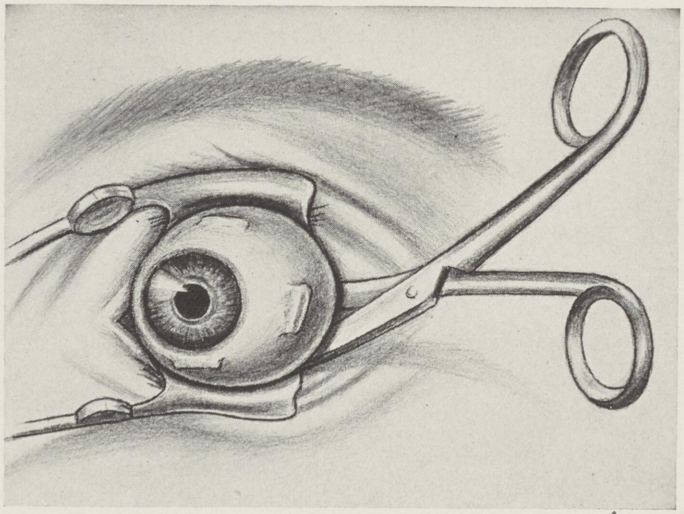


Fig. 81.

The sclera is then divided behind the plane of the ciliary body; this removes the anterior segment of the globe. The contents of the posterior segment are removed by spoon, curet or sponge. All remnants of the choroid must be curetted away with thoroughness, particularly in the region of the optic nerve, and the scleral sac must be mopped dry.

The wound is left open for drainage. The postoperative treatment is antiseptic and symptomatic. The local symptoms for which evisceration is indicated usually subside very rapidly after the operation. If no intracranial metastasis has occurred, the improvement is general.

CHAPTER IV

THE CONJUNCTIVA

ESSENTIALS OF THE ANATOMY

The conjunctiva is a mucous membrane which lines the lids and covers the anterior surface of the eyeball. Between the palpebral and bulbar divisions the conjunctiva lies in folds concentric with the orbital margin; these folds constitute the fornices: Superior, inferior, lateral and medial. The conjunctiva is firmly attached only around the cornea and over the tarsi (see Plates I and II). The entire membrane is called the conjunctival sac. The fornices form the conjunctival culdesac. The membrane is described in four parts.

The palpebral conjunctiva lines the eyelids. It extends from the gray line in front of the orifices of the ducts of the meibomian glands to the orbital borders of the tarsi. It is continuous with the lining of the excretory lacrimal passages. There are few mucous glands and no adenoid tissue in the zone lying between the gray line and the adjacent border of the tarsus, which is a point of clinical significance in trachoma. Mucous glands and adenoid tissue are found in the tarsal areas; what have been described as papillæ may be islets of thickened mucous membrane.

The conjunctiva of the fornix is loosely folded, which permits free rotations of the globe. By means of fibers from the sheaths of extra-ocular muscles, the fornix is retracted by contractions of those muscles. In this portion the tunica propria is more fully developed; there are more mucous glands and thicker adenoid tissue. White blood-corpuscles normally aggregate in this region. Lymphatic concentrations form into nodes which are probably identical with Henle's so-called trachoma glands.

The ocular or bulbar conjunctiva covers the anterior segment of the eyeball. It is subdivided into a scleral and a corneal portion. The former is thin and transparent so that the white sclera shows through. It is movable over the sclera because of the interposed episclera. The three tissues unite near the cornea. The conjunctival epithelium covers the cornea. The bulbar conjunctiva has few mucous glands and little adenoid tissue.

The fourth division is the plica semilunaris, which is considered in connection with the lacrimal apparatus.

The blood supply of the conjunctiva is derived from two sources. Arteries

surround each tarsal plate. Branches from these vessels supply the conjunctiva of the lids, of the fornix, and of the globe to within 4 or 5 millimeters of the cornea. These are the posterior conjunctival vessels. One or two branches from the ophthalmic artery follow each rectus muscle. At the insertions of the tendons these branches divide into superficial or anterior conjunctival, and deep or anterior ciliary vessels.

The superficial twigs again divide; one set anastomoses with the posterior conjunctival terminals, while the other enters the episcleral tissue and ends at the margin of the cornea in a series of loops which constitute the corneal plexus. The deep branches perforate the sclera in front of the insertions of the muscles and enter the ciliary body. The point of perforation is marked by a dot of pigment (Fig. 82).

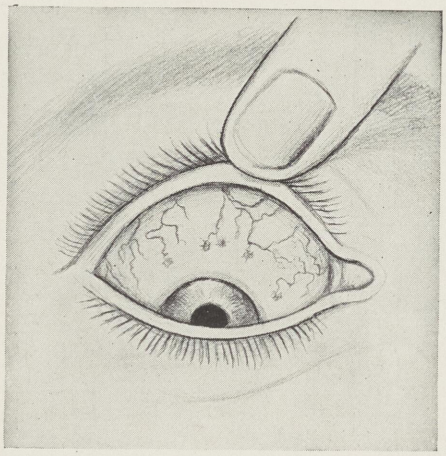


Fig. 82.
(After Dalrymple.)

The posterior conjunctival vessels, lying in the relatively loose bulbar conjunctiva, are comparatively coarse and move freely with the conjunctiva. Because of their rich anastomoses they are not obliterated by pressure. They terminate at 4 or 5 millimeters from the cornea. These points differentiate inflammations of the conjunctiva from those of deeper tissues. Being covered by only a few layers of cells, blood shows through their almost transparent walls with little alteration in color.

The episcleral vessels are only slightly movable, and anastomose less freely. When engorged they can be momentarily obliterated by pressure. They are more deeply covered, and the blood as seen through the tissues is pinkish. These vessels are engorged in superficial inflammations of the cornea.

The deepest or anterior ciliary vessels are immovable. They are disposed radially from the corneal margin for 2 to 4 millimeters and do not anastomose. They are obliterated by pressure. Being deeply covered, the blood as seen within them is purplish or violet. These vessels are engorged in inflammations of the deeper corneal layers, and of the iris and ciliary body. Diagnosis is facilitated by due attention to the origin, situation and behavior of the vessels in the three planes.

The veins of the conjunctiva generally follow the course of the arteries. The larger return is by way of the palpebral veins, while the remainder drains by way of the muscular branches into the ophthalmic veins.

The lymphatics are superficial and deep; both drain toward the commissures and unite with those that have been described in connection with the lids.

The nerves of the conjunctiva are general sensory, from the ophthalmic division of the trigeminus. The lacrimal supplies the lateral portion, and the infratrochlear supplies the medial. These terminate in free ends or in tactile corpuscles. The cornea is supplied by the anterior ciliary nerves.

INSPECTION OF THE CONJUNCTIVA

If the surgeon places his thumb below the lower lid and makes traction downward, and directs the patient to look upward, he can inspect the conjunctiva from the lid margin to the cornea. In examining the conjunctiva above, the patient is directed to look downward. The surgeon grasps the lashes and pulls the lid outward from the globe, and at the same time presses backward and downward with a probe or toothpick applied to the supratarsal skin fold; the lid is everted by rotating the lid margin toward the orbit over the probe. The eversion is maintained by pressure with the thumb that is engaged in grasping the lashes.

By adjusting the probe and again pressing downward with it, the upper fornix is exposed. It can be exposed in another way. While the upper lid is everted the patient is directed to look down; the examiner then slips the lower lid behind the upper, and the membrane of the fornix comes into view. To replace the upper lid it is pulled outward by the lashes and the patient is directed to look upward. The lateral or medial fornix is exposed by making traction on the skin temporally or nasally, while the patient looks in the opposite direction.

A child resists examination, particularly when the eyes are tender or painful. Where there is marked resistance against separating the lids with the fingers, it will be safer to use strabismus hooks. An attendant holds the child's body and limbs while the child's head is held face up on the surgeon's lap. The surgeon takes a lid hook in each hand, and at the same time holds the child's head. A hook is inserted beneath the upper lid at the outer canthus. That lid is slightly elevated and the other hook is inserted beneath the lower lid at the same point. By manipulating the hooks, the conjunctiva and cornea can be thoroughly inspected.

HYPEREMIA OF THE CONJUNCTIVA

Hyperemia of the conjunctiva exists independently, or combined with injection of the episcleral or ciliary vessels or both. When the injection does not approach the cornea by 4 millimeters, it is conjunctival hyperemia. When the

injection does not extend more than 4 millimeters from the cornea, it is ciliary hyperemia.

Adrenalin solution contracts the vessels more promptly and completely in the first condition than in the second. Secretion, when present, will be mucoid in the first instance, and watery in the second. Both systems of vessels may be engorged at once.

Hyperemia of the conjunctiva is due to mechanical causes, as dust; it is symptomatic as in eye-strain, or it is a complication as in hay-fever or coryza. The patient complains of smarting, burning, or itching sensations about the eyes, and lacrimation is increased.

Treatment.—Cold compresses for five minutes every hour are helpful in severe cases. The cause should be sought and removed. Refractive errors and muscle imbalance must be corrected. Camphor water 5 per cent in a saturated solution of boric acid, with 1:10,000 adrenalin chlorid, one drop in the eye every two hours is soothing and beneficial.

CONJUNCTIVITIS

Inflammations of the conjunctiva are usually classified as catarrhal, purulent, membranous, granular and phlyctenular. (See page 153.)

Catarrhal Conjunctivitis is subdivided into acute, chronic and follicular forms. In the acute form the conjunctiva is smooth and somewhat relaxed, of a uniformly red or velvety appearance, and considerable mucus is present. It is often present with acute respiratory infections and the exanthemata. It is especially constant in measles.

The secretions accumulate as flakes or shreds which act as irritating foreign bodies. Their presence in the lacrimal sac may excite a dacryocystitis. They may injure the corneal epithelium and produce an ulcer. They cause the lids to adhere together at night, and may excite a blepharitis. The lid borders are usually swollen. Iritis may develop.

The flakes produce the sensation of a foreign body, with itching, burning and lacrimation. Pain is present when the cornea is involved. Photophobia may be marked. If the secretion becomes mucopurulent the symptoms increase in severity. The instillation of a sterile 1 per cent solution of methylene-blue will be helpful in demonstrating the flakes and shreds.

The disease persists for one or two weeks. It may become chronic if the cause is not abated, and then the conjunctiva will show the effects of prolonged inflammation. The short duration is characteristic for diagnosis. Rarely pustules will develop near the corneal margin, and these may be confused with phlyctenular conjunctivitis.

Treatment.—Permit free drainage of the secretions; never use a bandage! Remove the cause. The medical treatment is symptomatic, as no specific organism is responsible. The eyes should be protected with dark glasses. Cold compresses are applied for five minutes at intervals of one to four hours. Instill one or two drops of 25 to 50 per cent solution of argyrol, and after a few minutes irrigate the conjunctival sac with warm boric acid solution. Argyrol coagulates the accumulated secretions which can be removed by irrigation; this prevents contamination of the lacrimal sac. To prescribe argyrol for the patient to use without supervision invites local argyrosis.

A good effect is obtained by painting the surfaces with fresh 2 per cent solution of silver nitrate. A wooden toothpick is tightly wound on each end with cotton; one end is saturated with the silver solution, and the other with normal salt solution. The lids are everted; each is painted lightly with the silver, and when a film resembling skimmed milk has formed, it is neutralized with the salt solution.

Zinc sulphate is useful; one drop of 0.25 per cent solution is instilled into each eye three or four times a day. The following is a useful prescription:

℞	Zinc Sulph.	0.016	Gm.	(Gr.$\frac{1}{4}$)
	Phenacain Hydrochl.	0.065	Gm.	(Gr.j)
	Aq. Camph.	0.4	c. c.	(min.vj)
	Adrenal. Chlor. (1:1,000) ..	0.678	c. c.	(min.x)
	Acid. Bor................	0.25	Gm.	(Gr.iv)
	Aq. Dest. q. s. ad	7.8	c. c.	(ʒij)

M. et filt. Sig. One drop in each eye every 2 hours.

White's ointment should be used at night; it has a good effect on the disease, and tends to prevent adhesions between the lids from the drying of the secretions upon them.

The patient must be impressed with the contagious character of the disease, and the necessity of protecting those associated with him at home or at work. Each patient should have individual linen and basins. Many organisms have been identified in different cases presenting similar clinical pictures. The pneumococcus, Koch-Weeks bacillus, Morax-Axenfeld diplobacillus, *Micrococcus catarrhalis* and staphylococcus are the organisms most frequently identified with catarrhal conjunctivitis.

After the disease subsides it is well to refract the cases that show an inclination toward recurrences.

Epidemic Conjunctivitis, or "pink eye," is caused by the pneumococcus or the Koch-Weeks bacillus, and is highly contagious. Each produces marked reactions in the tissues. The former infection lasts from eight to ten days,

and the latter from one to two weeks. The *Micrococcus catarrhalis,* which is not to be confused with the gonococcus, and the staphylococcus are also causes of epidemic conjunctivitis. Differentiation is only possible with the microscope. The treatment for these is by methods already described.

Angular Conjunctivitis is caused by the Morax-Axenfeld diplobacillus, and is so named because its tendency is to affect the canthal angles of the conjunctival sac. It is persistent and prone to become chronic. Zinc sulphate in 0.25 to 1 per cent solutions acts as a specific. One drop of the solution is to be instilled every four hours, and an ointment of the same strength is used at bedtime.

Other acute types of catarrhal conjunctivitis complicate certain of the acute diseases, such as influenza and meningitis. In these cases the organism belongs to the major disease.

Mercurochrome 220 may be used in 1 or 2 per cent solution.

Ethylhydrocuprein hydrochlorid, or optochin, in 1 or 2 per cent solution, is of use in the form due to the pneumococcus; some reports have indicated ill effects on corneal tissue coincident with its use.

After the acute symptoms have subsided, it is advisable to use a mild astringent solution. The following are useful formulas:

	℞	Zinc. Sulph.	0.03	Gm.	(Gr.ss)
		Acid. Bor. } aa Sod.Bibor. }	0.3	Gm.	(Gr.iv)
		Aq. Dest. q. s. ad	7.8	c. c.	(ʒij)
M. et filt.	Sig.	One drop in each eye every 4 hours.			
	℞	Acid. Bor.	0.3	Gm.	(Gr.iv)
		Aq. Camph.	0.8	c. c.	(Gtt.xij)
		Sol. Hydrarg. Chlor.			
		Corr. 1:10,000 q. s. ad	7.8	c. c.	(ʒij)
M. et filt.	Sig.	One drop in each eye every 4 hours.			

Water miscible fluid hydrastis up to 15 per cent may be added to either formula.

Newer forms of conjunctivitis have been recognized, and are designated descriptively:

Swimming Bath Conjunctivitis occurs in mild epidemics among those who frequent a certain pool.

Agricultural Conjunctivitis occurs among those who farm in a district where the disease prevails. In this form lymph drainage is retarded, as is shown by swollen lids and enlarged glands.

Squirrel Plague Conjunctivitis is caused by the *Bacterium tularense;* it is the oculoglandular manifestation of tularemia. It occurs among those who

handle infected rabbits. The eye is irritated and the lids are swollen. Lacrimation is moderate to profuse. There is localized redness and chemosis of the palpebral or bulbar conjunctiva; this is followed by discrete ulceration with a punched-out appearance. The regional lymph glands are swollen, tender and painful. Constitutional symptoms are present.

The diagnosis is made from the history of handling infected animals, the course of the disease, and by the agglutination tests. It must be differentiated from Parinaud's conjunctivitis, Mikulicz's disease, and sporotrichosis of the conjunctiva; it might be confused with von Recklinghausen's disease. The treatment is rest in bed, and symptomatic. When the affected glands suppurate, they should be incised.

Follicular Conjunctivitis is characterized by light-colored elevations on the conjunctiva of the fornices and tarsi. The elevations are often arranged like rows of beads. Mild cases will recover spontaneously, and severer ones yield to such treatment as has been outlined. Recurrences suggest the presence of refractive errors.

The character of the elevations serves to confuse the disease with trachoma, from which it is impossible, oftentimes, to differentiate it in the early stage. Where the disease does not disappear promptly, or at most after two or three weeks, it must be regarded as trachoma and treated as such.

Complications of catarrhal conjunctivitis are ulceration of the cornea, iritis, blepharitis, and ectropion. The cornea is abraded by the shreds and rough flakes of coagulated secretion, especially after the eye has been bandaged; these abrasions usually heal promptly. Iritis is due to endogenous infection. Blepharitis is caused by the secretion continually flowing over the lid margin, and ectropion arises in consequence of severe and prolonged blepharitis.

PURULENT CONJUNCTIVITIS

Purulent conjunctivitis is caused by the gonococcus, and less frequently by the *Micrococcus catarrhalis* and the meningococcus. The clinical behavior is similar, but much more severe in the first named. To differentiate them culturing is necessary. Practically all such cases should be regarded and treated as gonococcal infection. Three epochal forms of gonorrheal ophthalmitis are designated as ophthalmia neonatorum in the newborn, postnatal in infancy, and adult, which comprises all cases occurring after the period of infancy.

Ophthalmia Neonatorum is comparatively rare because of the prophylactic treatment known as Credé's method. This consists of gentle but thorough cleansing of the lids, and the instillation of one drop of a 2 per cent solution of nitrate of silver into the conjunctival sac of each eye; the silver need not be

neutralized. Although attempts have been made to substitute solutions of the newer salts for the nitrate, they have demonstrated no superiority.

After infection has occurred, the period of incubation varies from one to three days; when the organism has low virulence it may be four days. Any development after the fourth day is to be regarded as a postnatal infection for which careless handling or incompetent nursing is responsible. The management of the patient disregards the time at which infection occurred.

The earliest sign of the disease is a moderate edema which smooths out the wrinkles and folds of the skin of the lids. The edema increases and the skin shows evidence of inflammation; the conjunctiva is inflamed, and the eye is tender and painful.

Pressure increases the possibilities of damage. It is better to separate the lids with strabismus hooks in making the examination; the conjunctival sac can be cleansed, and appropriate treatment can be applied with the one preparation.

The conjunctiva is swollen and uneven, or almost nodular; in less severe forms it is smooth, even though thickened. As the infiltration in the lid tissues progresses, the bulbar conjunctiva is invaded, and in much the same manner. The infiltration extends to the margin of the cornea only; the conjunctiva is markedly chemotic. The cornea has the appearance of being recessed beneath the plane of the conjunctiva. The secretion is serosanguineous and is mixed with coagula of pus. Fever and lymphatic involvement indicate constitutional reactions.

Treatment.—Owing to the increased local heat, cold or iced compresses can be used for five minutes in each quarter, half or one hour, night and day with safety. This is more frequent than can be recommended in milder diseases. Four to six times in twenty-four hours the eye should be irrigated with warm boric acid solution, or with solution of potassium permanganate, beginning with 1:10,000, and gradually increasing to 1:5,000.

Investigations have demonstrated that the organisms grow between the superficial epithelial cells of the conjunctiva and invade the deeper layers. Also that the younger epithelial cells have phagocytic activity. Consequently it is desirable to remove the more superficial cells as they are devitalized. Potassium permanganate is peculiarly adapted to such a purpose, but must be first used in low dilutions.

One drop of silver nitrate 1 per cent solution is instilled about four times a day during the severe stage. These methods may prevent the active pus stage of the disease, or modify its severity. In this stage the secretion becomes more abundant and thickened in consistency, like a creamy liquid tinged with yellow or green.

The duration of each stage is two days or more. Where the cornea has not become involved, the course of the disease tends toward recovery. The swelling subsides, the secretion diminishes, the pain and tenderness are relieved, and the tissues gradually resume a normal appearance.

Where the disease assumes a more severe character and complications arise, the course will be prolonged over weeks or perhaps months. The swelling of the lids may attain such proportions that it is impossible to separate them sufficiently for adequate examination and treatment. In such a situation an external canthotomy must be made. This is advisable in ulceration of the cornea where there is a prospect of perforation. The pressure exerted by contractions of the orbicularis can result in disaster, and the muscle is weakened by the operation.

Convalescence may be slow, and in such patients the disease may be regarded as chronic. Cleansing of the conjunctival sac and the continued use of *fresh* solutions of silver is indicated. Mercurochrome 220 in 2 per cent solution has been used in all forms and in all stages of gonorrheal ophthalmia.

Hygiene and prophylaxis are imperative. Credé's method is employed immediately after the birth of the child. If an inflammation appears in one eye, the other should be treated with silver nitrate as in the first instance, and protected from contamination by secretions from the affected eye. This is accomplished with a Buller's shield (see Fig. 51).

A watch crystal or piece of celluloid is fitted over the orbital opening, and sealed in place with adhesive strips to the brow, nose, cheek and temple. This sealing is made as complete as possible except at the temporal side, where an opening is left for ventilation. The patient is to occupy a position in which it is impossible for the secretions from the affected eye to flow toward the sound eye. The transparent shield permits constant inspection of the unaffected eye.

All dressings used about the patient's eyes must be dropped into an antiseptic solution at once, and later burned. Instruments, syringes and medicine droppers should be sterilized after use and kept in an antiseptic solution. Bed and personal linen should be changed daily or as soon as soiled, and placed in disinfectant.

The attendants wear gowns and gloves while treating the patient, and should thoroughly cleanse their hands immediately after the treatment. They should wear glasses. If secretion from the eye of the patient spurts into the eye of the surgeon or nurse, a drop or two of 50 per cent solution of argyrol should be instilled at once to coagulate the secretions. After the lapse of a few minutes the coagula are removed by irrigation with warm boric acid or normal salt solution. Any further treatment will depend upon the reaction.

Prognosis is always grave; the amount of vision retained is dependent

upon corneal involvement or intra-ocular changes. The cornea is recessed below the level of the surrounding chemotic conjunctiva. Secretions will collect in the depression, and care must be exercised to thoroughly remove them by very gentle irrigation. The chemosis also tends to obstruct the circulation in the arterial loops that encircle the cornea, and this produces stasis. Thus the cornea may become involved by direct infection or by interference with nutrition.

Direct infection is manifested by the formation of ulcers. When the cornea is perforated the aqueous escapes and the iris prolapses into the opening. It adheres to the cornea and is transformed into connective tissue where incarcerated; an adherent leukoma results. With loss of nutrition, the whole cornea breaks down and sloughing occurs. The usual outcome is panophthalmitis with collapse, shrinking, or loss of the globe. Should perforation occur in the pupillary area of the cornea, the lens, and not the iris, will come forward and be expelled or seal the opening.

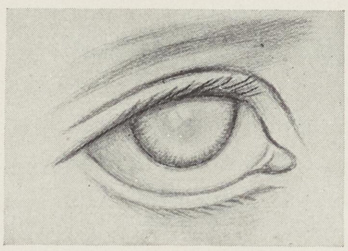


Fig. 83.

Should the lens stop the aperture, its capsule will adhere to the posterior surface of the cornea (Fig. 83). The anterior chamber will have been obliterated from the loss of aqueous. While the cornea undergoes repair the lens epithelium grows between the cortex and the new tissue. When the anterior chamber re-forms the lens will be torn from the cornea. The site of adhesion is marked by a dense lens opacity; this is an acquired anterior polar cataract. The cornea may regain transparency but the lens does not. The obliteration of the anterior chamber causes a secondary glaucoma. The eyeball enlarges uniformly, and if the cornea becomes clear the eye will have the appearance of buphthalmos with cataract.

Some cases will be complicated by intra-ocular inflammation, as iritis or iridocyclitis. It is good practice to instill one drop of 1 per cent solution of atropin sulphate into the eye twice daily.

Inasmuch as organisms other than the gonococcus may be responsible for the disease, no surgeon should commit himself as to the nature of the causative agent without laboratory support. Medicolegal and domestic complications are important possibilities.

Postnatal Gonorrheal Ophthalmia differs only in that the infection occurs later.

Gonorrheal Conjunctivitis in the Adult presents no important differences from that in the newborn, so far as diagnosis and treatment are concerned. The mode of infection is by the fingers or by the use of contaminated linen.

The disease does not immunize the individual, and a person may convey the infection from the genitalia to the eyes. The prognosis is said to be more grave in the adult than in the infant.

Silver is to be used only while there is secretion. When the lids can be everted they should be painted with a 1 or 2 per cent solution of silver nitrate, as in catarrhal conjunctivitis. The excess may be neutralized or not. Irrigations should be carried out as in the case of infants. The pericorneal chemosis may be treated by several incisions radiating from the cornea. A cataract knife is best for this purpose; the back of the knife faces the cornea, the point is introduced into the corneal margin of the conjunctiva, and as it is advanced the knife cuts its way out.

Girls of two to ten years who have a purulent vaginal discharge are frequent victims of a purulent conjunctivitis. This is rarely of gonorrheal origin. These cases are quite amenable to treatment.

Metastatic Gonorrheal Conjunctivitis is due to infection conveyed by the blood stream. It is usually mild, but rather chronic in duration. Serum therapy is theoretically indicated in this type. As the disease confers no immunity, it has not apparently benefited by such treatment in other types. Nonspecific protein therapy is advocated by many surgeons. The parenteral injection of 5 to 10 cubic centimeters of partially sterilized milk every three or four days has given good results. Antityphoid vaccine and antitetanic serum have also been used. Such benefits as accrue are believed to be due to a stimulation of leukocytosis.

MEMBRANOUS CONJUNCTIVITIS

Membranous conjunctivitis is characterized by the formation of a membrane that extends into the subconjunctival tissues when true or deep, or that lies upon and is loosely attached to the conjunctiva when false or superficial.

It may be caused by any one of several organisms: The gonococcus, pneumococcus, meningococcus, streptococcus, Koch-Weeks bacillus or the Klebs-Löffler bacillus. Cultures and inoculations are required for differential diagnosis, as clinical evidence is insufficient. The amount and character of secretion may be suggestive, but they are not diagnostic. The importance of a positive identification can hardly be overemphasized.

When we designate "membranous" conjunctivitis, we allude to either of two forms: the true diphtheritic form which is caused by the Klebs-Löffler bacillus; or the nondiphtheritic or diphtheroid form which is caused by any organism that produces similar changes within or upon the tissues.

The virulence of the organism and the defense capacity of the host will determine the extent of the damage. Where the virulence is high or the

defense inadequate for the situation, the disease will extend deeply into the tissues. Where the virulence is low and the defensive forces are competent and active, the effects will be limited to superficial manifestations. Consequently, a wide latitude of symptomatology may be expected.

The disease is not very prevalent, but because of possible epidemics and fatalities, the management of such cases enjoins an important responsibility. The secretions from these cases must be presumed to be infectious until they are proven innocuous. All of the injunctions recited under purulent conjunctivitis are pertinent here, and in addition, the patient must be *isolated*.

Diphtheritic Conjunctivitis is more especially a disease of childhood. The signs of an intense inflammation may be expected. The conjunctiva is subject to no more severe involvement than that produced by the Klebs-Löffler bacillus. The lids are not only thickened, but stiff, so that it may be almost or quite impossible to expose the palpebral conjunctiva.

The thickening is caused by the coagulation of an exudate which fails to reach the surface. Because of this coagulation, secretion is not abundant; the tissues are clogged by the exudate, nutrition cannot be maintained, and some necrosis occurs. The compression of the vascular supply interferes with the nutrition of the cornea, and it may slough. The perivascular lymph channels are compressed, and the preauricular and submaxillary glands are enlarged. The conjunctiva has the appearance of a diphtheritic membrane as seen in the nasopharynx. This surface may not be uniform in depth or regular in outline.

Duration is from seven to ten days, depending upon the depth of the infiltration. An increase of secretion is noted as absorption begins; devitalized tissue will slough, and granulations will make their appearance. The secretion increases in volume, and cicatrization begins. Where granulations appear on opposing surfaces of the bulbar and palpebral conjunctiva, adhesions or symblepharon will probably form. Small pledgets of cotton impregnated with sterile petrolatum and placed in the conjunctival culdesacs may thwart this tendency. Cicatrization is attended by contraction, and this may reduce the size of the conjunctival sac. It may also produce entropion and trichiasis.

Occasionally the coagulated exudate stimulates an exuberant proliferation of connective tissue. Microscopical sections cut from excised portions of the hypertrophied structures show masses of fine fibers that have no definite arrangement. They contain no nerve-fibers. Recovery is very slow. The excess tissue can be pared away or excised, often without anesthesia. The normal conjunctiva no longer exists.

Superficial diphtheritic conjunctivitis differs from the preceding in degree and severity. The exudate reaches the surface, where it is coagulated in the

form of a loose membrane. It can be removed readily but without benefit, as it quickly re-forms. The secretion is scanty at first; later it increases in amount and becomes catarrhal rather than purulent. Although the cornea may be affected, it seldom sloughs. In this form the conjunctiva is restored in nearly all cases.

Treatment is constitutional and local. Diphtheria is attended by profound prostration. Supportive treatment is indicated. The disease is amenable to antitoxin. This should be given in all serious and doubtful cases. The initial dose is repeated in twelve hours, and as often thereafter as seems expedient. Because of the vascular compression, hot applications should be used to favor dilatation of the capillaries.

Irrigations with 1: 6,000 solution of bichlorid of mercury or potassium permanganate are useful. One per cent solution of quinin sulphate by instillation is said to be effective, but it should contain the minimum quantity of acid that will dissolve the salt. In the lighter forms, after the membrane has separated, Fuchs suggests painting the conjunctiva with 1: 1,000 solution of bichlorid of mercury.

After the secretion has become well established, 1 per cent solution of silver nitrate is of value when applied as for purulent conjunctivitis. When it irritates, it is to be discontinued. The treatment of corneal complications is discussed in the proper section. Instill one drop of a 1 per cent solution of atropin sulphate twice daily.

Paralysis of accommodation without or with impairment of pupillary activity may occur, and no treatment has as yet seemed to hasten recovery. It disappears after several months.

Pseudodiphtheritic or nondiphtheritic membranous conjunctivitis occurs usually as a complication to some general disease. When it is due to the streptococcus, especially if coincidental to one of the exanthemata, it is apt to be severe, and to have direful consequences. Blindness or death may result.

GRANULAR CONJUNCTIVITIS OR TRACHOMA

Granular Conjunctivitis (Trachoma) is called *granulated lids* by the laity, because follicles or granulations are studded over the inflamed conjunctiva. But the laity uses the term without discrimination.

This is an inflammatory disease that affects the palpebral conjunctiva principally; it is characterized by profound hyperplasia which is followed by atrophy. The tarsus is deformed by the process, and the misshapen tarsus is a mechanical cause of corneal complications. Trachoma is transmitted by infection, but no one organism has ever been proven to be respon-

sible.* There is usually more or less purulent secretion in every active case. This may be due to an intercurrent or complicating infection, especially in so-called acute cases.

The disease occurs regardless of country, climate, season, altitude or race. The exemption of the Negro is questioned. The United States admits no immigrant who has the disease, and where a question of diagnosis exists, the presumption is against the applicant.

Trachoma spreads most rapidly where people are crowded into close quarters, as in jails, asylums, army barracks and tenements, and among those of the poorer and more ignorant classes. When Napoleon conducted military operations in Egypt, about three-fourths of his army was invalidated because of ophthalmia; the contrast in temperature between the intense heat of the day and the sharp chill of the night was believed to be the cause of the disease. It is significant that the chill night air would prompt the soldiers to keep closely huddled together.

Egyptians, particularly the poorer classes, are rarely found free from trachoma. Their religion forbids them to kill except for food; flies swarm unmolested about the eyes of the infected and uninfected alike. The American Indian is very susceptible. Among the white population of the United States the disease is particularly prevalent in West Virginia, Tennessee, Kentucky and in southern Illinois. Negroes possess a high degree of immunity.

Trachoma invades the conjunctiva of the fornix, the tarsal area in the upper lid especially, and the inner canthus about the caruncle. It seldom directly attacks the bulbar conjunctiva. From the inner canthus the infection occasionally invades the lacrimal sac and excites a dacryocystitis. Seldom is the disease limited to one eye.

Adenoid tissue is apparently required, for a trachoma-free zone exists between the ciliary border of the tarsal plate and the free border of the lid. The upper lid is affected much more frequently than the lower. In follicular conjunctivitis the lower lid is more readily affected. Both lids must be examined.

Clinically, there are three forms of trachoma. The *papillary* form is limited to the tarsal area, and in appearance somewhat resembles the surface of a ripe red raspberry. Rounded elevations are separated by clefts or fissures. Because of the hypertrophy of the membrane the meibomian glands cannot be seen. The *granular* form is more generally distributed, and is particularly evident in the folds of the fornices. In this situation the follicles suggest rows of sago grains, soft and gelatinous, but reddened. The adenoid layers of the

* Noguchi recently announced that he had isolated an organism from selected cases of trachoma, and that with pure cultures of this organism he had produced a trachoma-like disease in monkeys.

conjunctiva are deeply invaded. The *mixed* form is a combination of the preceding two.

To these may be added a final form in which atrophy of the membrane, destruction of the adenoid tissue, deformity of the tarsus and cicatricial contractions of the stroma of the conjunctiva have occurred. The palpebral aperture is shortened and narrowed, there is an appearance of ptosis, and the expression is one of drowsiness. While the bulbar conjunctiva may not be invaded by the disease, there is considerable dilatation of its blood-vessels; the membrane is thickened from friction with the lids, but it rarely contains true granulations.

Papillæ or granulations consist of compact masses of round cells enveloped in connective tissue, and covered on the exposed surface with thickened and hardened layers of epithelium. These elevations are rough, and they scratch the cornea as the lid moves over it. This irritation accounts for the presence of lacrimation, photophobia and blepharospasm. The continued irritation stimulates an effort toward repair, and pannus is formed. The same mechanical effects may be due to a deformed tarsus or cicatricial bands.

Granulations in trachoma and in granulating wounds have nothing in common except the name and appearance; the process in each case is distinct. Where the granulations are especially harsh, the injury to the cornea will be more profound. When a complicating infection is added, or the nutrition of the cornea suffers unduly, ulcers will occur on that structure after the epithelium has been destroyed down to Bowman's membrane.

Since an ulcer of the cornea calls for protection and repair, blood-vessels make their way from the nearest point to the ulcer. In this manner *fascicular pannus* is formed. It is concerned with healing the ulcer. It will be multiple where ulcers are numerous or successive (see Fig. 109-B).

True trachomatous pannus belongs to trachoma only. It practically always forms from above, where the lid and cornea are in almost constant contact. The epithelium of the bulbar conjunctiva and cornea is lacerated by the granulations, and secretion is present. A new membrane, rich in blood-vessels, grows in between the epithelium and Bowman's membrane; it has a roughened appearance somewhat similar to that of the lids. That the lid is of importance in the production of pannus is suggested by the advancing edge of the latter, which is usually parallel with the lid border.

Trachoma may develop so insidiously that it does not attract attention to its true nature for a long time; or it may quickly command consideration because of lacrimation, photophobia and pain. In the latter case there is a thickened aspect of the skin of the lids due to blepharospasm. Where photophobia exists it is well to watch for the complication of iritis.

Should the symptoms become particularly intense, an intercurrent conjunctival infection is probably responsible; the presence of granules over the tarsi or in the fornices will excite suspicion. Granules may not be in evidence, but they will appear in due time if it is trachoma. They are often concealed by the hypertrophy of the membrane. Where granulations are present, the disease should be treated as trachoma, although it may be a severe follicular conjunctivitis; this therapeutic or duration test may afford the only means of determining the true character of the disease.

When the secretion is abundant and purulent, it is sometimes impossible to distinguish trachoma from gonorrheal conjunctivitis without a microscopical examination of the secretion; when in doubt this should be made. The gonococcus should be sought in several smears. If the secretion is free of the organism, gentle scrapings of conjunctival epithelium may expose it. Scraping is done with a sterile platinum loop, the end of which has been flattened like a stirrup. Where identifying organisms are not found and the disease persists more than two weeks, it is probably trachoma.

The *symptoms* of trachoma are rather constant in character but variable in degree. The course is characterized by remissions, intermissions, relapses and exacerbations. It is a chronic disorder, the tissue changes are progressive, and recovery in the true sense is doubtful in any except the mild cases. Pain, photophobia and lacrimation vary according to the degree of irritation present in the conjunctiva and cornea. Vision varies according to the involvement of the cornea by pannus and ulceration. Secretion varies from profuse, in the stage of hypertrophy, to scant or absent in the stage of atrophy and shrinking.

The degree of disability depends upon the severity of the symptoms regardless of the stage of the disease. After a duration of years, economic disability may become total and permanent.

In many cases light is an irritant and excites violent sneezing. It is probably due to irritability of the corneal nerves. Cocainization of the cornea or of the sphenopalatine ganglion has little or no effect, but ointments modify the symptom favorably.

Cicatrization involves the tarsus. As the conjunctiva shrinks, it deepens the posterior concavity of the tarsus. This rotates the free border of the lid inward and produces trichiasis and entropion. It also rounds off the angle on the posterior margin of the free border of the lid. An ectropion may form from the intense blepharospasm (Plate III). The free borders of the lids are usually thickened and this may produce trichiasis without tarsal deformity. The contractions of cicatrization, together with atrophy, in many cases obliterate the folds of the fornices and reduce the size of the conjunctival sac.

Xerophthalmia or xerosis is a condition in which the atrophy has destroyed

PLATE III.—CROSS-SECTION OF UPPER EYELID FROM TRACHOMA.

The orbicularis oculi and tarsus are hypertrophied. Ectropion from deformity of tarsus. *CT*, connective tissue; *N*, nodule. Round-celled infiltration. (Compare with Plate II.)

the conjunctival mucous glands. The secretion is foamy or frothy in character; tears will neither mix with it nor adhere to the glazed surface. Keratinization of the corneal epithelium occurs. The process continues until the conjunctival culdesac is so contracted that the lids can no longer close over the globe; the cornea is exposed and becomes opaque. The lacrimal gland atrophies. The condition resembles a total symblepharon. The patient has a sensation of intolerable dryness. The treatment is palliative and consists of phenacain 1 per cent in ointment.

Trachoma is communicated from one eye to another by the secretion, and the patient must protect his associates. Individual basins and linen are required. Secondary or intercurrent infections must have prompt attention, for the more profuse the secretions, the more readily are others infected.

No treatment is specific, and there is no uniform success with any one method. While hypertrophy is present and secretion is abundant, 2 per cent solution of silver nitrate daily is indicated. The conjunctiva is cocainized, and the lids are everted and treated according to the method described under catarrhal conjunctivitis. This has to be continued over a considerable period of time, and no solution more than a week old should be used.

Some surgeons have reported good effects from silver by increasing the strength from day to day, but not above 10 per cent, until the reaction is quite severe. By double eversion of the lids the fornices are exposed, and it is necessary to treat this part of the membrane in every case except the pure papillary form. The treatment should be designed to control the disease as quickly as possible in order to minimize the contractions of the conjunctiva.

Copper sulphate is indicated when the secretion diminishes and the hypertrophy of the membranes persists. It is contra-indicated in ulceration of the cornea, in active inflammation of the conjunctiva, and in the cicatricial stage because it stimulates the proliferation of connective tissue. The pure stick is used daily, after the instillation of cocain. The reaction is severe, and iced compresses may be required following each treatment.

As the hypertrophy diminishes, the treatments are given each second or third day according to conditions. When the progress is satisfactory, applications of pure alum may be substituted for the copper, and the patient is given a 5 to 10 per cent ointment of copper citrate to use at home.

One of the older prescriptions is a 10 per cent solution of copper sulphate in glycerin. On the first day one drop of solution is added to nineteen drops of water; a drop of this is instilled into each eye four times that day. On the second day two drops of solution to eighteen of water are prepared and used as before. Continue to mix one drop more of solution and one drop less of water than was used on the preceding day, until the patient's tolerance

is reached and the reaction becomes too severe. The combination always totals twenty drops; it is necessary to mix it fresh each day.

Some have had success by patiently opening each follicle or granulation with the point of a knife, and then scrubbing the conjunctiva with a 1:1,000 to 1:500 solution of mercuric chlorid. A toothbrush with stiff bristles is used. This is grattage; it may require a general anesthetic, and postoperative use of iced compresses. A favorable reaction is sometimes secured by lightly scoring the conjunctiva a number of times parallel with the lid border and from one canthus to the other. This is done to open the follicles.

Knapp's roller forceps (Fig. 84) are used to break the follicles. The patient is anesthetized and the lid is everted. The forceps are applied, one roller in the fornix and the other on the conjunctival surface near the lashes. As the rollers are pressed together and pulled off the lid, the contents of the follicles are expressed. The effect is the same as though the doubled conjunctiva had been pulled between the rollers. The forceps are applied repeatedly until the entire conjunctiva of both lids has been treated. Where the canthi cannot be rolled satisfactorily, that region can be similarly treated with Noyes' forceps. Iced compresses are indicated until the reaction subsides.

Fig. 84.

An ointment of 0.25 to 0.5 per cent of zinc sulphate may be used at night. It has a good effect on the disease and prevents the lids from adhering together. White's ointment is used similarly.

Medical treatment must be continued over long periods, because the disease is prone to relapse. Continual vigilance is necessary.

Trichiasis, entropion and ectropion are to be treated operatively according to methods already discussed. Tarsectomy is to be considered where the deformed tarsus irritates the cornea; it is often combined with canthoplasty. The latter operation may be indicated at any stage of the disease, particularly for blepharospasm and lid pressure on the globe.

Pannus rarely needs individual treatment, as it generally disappears when the conjunctival irritation subsides. A variety of procedures have been proposed for cases that require attention. When an iritis or a corneal ulcer accompanies the pannus, one drop of 1 per cent solution of atropin sulphate is used daily. Ulcers usually respond to the treatment given the trachoma; they are nearly always shallow. They are not to be touched with copper; the eye should not be bandaged, but kept as free of secretions as possible.

Some eyes are sensitive to atropin, and some patients have made notable improvement when atropin was discontinued and mild collyria used.

Small, shallow and very painful ulcers have been observed to form intermittently in the terminals of the vessels composing a pannus. These are from septic emboli, and are caused by focal infections. It is necessary to locate and eradicate the source.

When treating a case of trachoma, the surgeon must shield his own eyes from infection, and should thoroughly cleanse his hands after every treatment. The treatment for accidental inoculation is given under gonorrheal conjunctivitis.

SPECIAL FORMS OF CONJUNCTIVITIS

Parinaud's Conjunctivitis has a local resemblance to trachoma. It develops with fever and other evidences of systemic disturbance, such as occur in the exanthemata. Usually but one eye is affected. On the same side the preauricular gland is enlarged, and the glands down the neck are frequently involved. Its duration is from one to five months. The local treatment is like that for trachoma in the stage of active secretion.

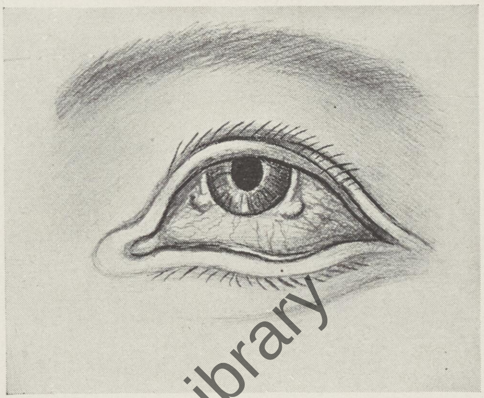


Fig. 85.—Phlyctenular Conjunctivitis. (After Parsons.)

Phlyctenular Conjunctivitis and phlyctenular keratitis (*q.v.*) are also called eczematous conjunctivitis and eczematous keratitis. The disease seldom affects the palpebral conjunctiva, but appears on the bulbar membrane and on the cornea. It often follows one of the exanthemata. It is prone to occur in children who are below par physically. Children with adenoids or swollen cervical glands are susceptible, and this justified the older name of scrofulous ophthalmia.

Grayish or yellowish elevations 1 to 3 millimeters in diameter appear discretely upon the conjunctiva, the cornea and the sclerocorneal junction (Fig. 85). They may appear in all of these situations simultaneously. The elevations consist of accumulations of leukocytes *beneath the epithelium*. They have no connective-tissue capsule as in trachoma. The apex of the elevation ulcerates, and the phlyctenule breaks down. Blood-vessels enter the area and form a pinkish zone about the base, which is in contrast to the color before ulceration.

When the disease is limited to the conjunctiva, the symptoms are not especially severe. One or 2 per cent of yellow oxid of mercury ointment is used

locally with gentle massage three times daily. The general condition may require treatment, or dietetic measures may suffice. Sweets and fried foods are forbidden. Outdoor exercise is encouraged. This is all that is required in the majority of cases (see phlyctenular keratitis).

Vernal Conjunctivitis or spring catarrh is a chronic, usually bilateral inflammation of the conjunctiva that is at its maximum in hot weather and at its minimum in cold. It is not accompanied by any constant organism. It has been mistaken for trachoma.

The elevations on the palpebral conjunctiva are flat and much broader than those found in trachoma. The clefts between them are steeper and deeper, and this gives an appearance of a cobblestone pavement (Fig. 86). The color is lighter, and the whole area seems to be covered with a bluish-gray or milky film. The bulbar conjunctiva, where exposed by the open lids, may be affected. Nodules form around the cornea; these resemble trachoma nodules, but are lighter in color. The bulbar and palpebral conjunctiva may both be affected at one time.

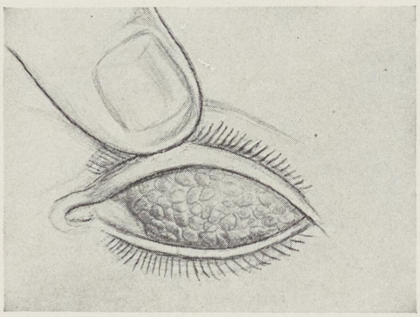


Fig. 86.—Vernal Conjunctivitis. (After Fuchs.)

The subjective symptoms are pain, lacrimation, photophobia, and itching of an intense character when the disease is in exacerbation. These subside when the disease is declining. The duration is from six to fifteen years.

Vernal conjunctivitis may be allied to hay-fever, although respiratory symptoms are only incidental accompaniments. It may be a protein sensitive manifestation, yet hot weather seems to be the one essential element to an aggravation of the disease. Eosinophils are constantly found in the tissues and in the secretions.

The treatment is palliative mostly. For the itching, Parsons recommends a solution of one drop of acetic acid in 15 cubic centimeters ($\frac{1}{2}$ fluid ounce) of distilled water to be used as an eye bath. Adrenalin chlorid solution 1 : 10,000 and weak solutions of zinc sulphate are useful. Dionin in 3 to 5 per cent solutions or ointment has given considerable relief. Radium has been used in recent years with much encouragement. The patient should wear smoked or tinted glasses and must avoid heat. Cold baths are soothing. Removal to a cooler climate has a salutary effect.

Simple Conjunctivitis is associated with various acute exanthemata, but is not likely to attain serious proportions unless complicated with streptococcal infection. Measles is one of the worst offenders. Acne is often accompanied by a conjunctivitis and a blepharitis of long duration.

Pemphigus of the Conjunctiva or essential shrinking of the conjunctiva exists separately or associated with similar lesions in the upper respiratory tract. The membrane is reddened, with white or gray spots which cover ulcerative processes that include the whole thickness of the conjunctiva. Shrinking occurs, and the manifestations resemble xerosis in the late stages. Similar treatment is indicated. Bullæ are not formed as in the skin, for the epithelium of the conjunctiva will not resist elevation like that of the skin. Hence rupture occurs, and the raw surface is covered with the gray film.

Lupus of the Conjunctiva extends to it from the skin of the lids.

When a conjunctivitis appears while chrysarobin is being used, the drug should be discontinued. An inflammation of the conjunctiva confined to the lower lid may arouse suspicion of the *purposeful introduction of some irritant.*

Atropin may excite a conjunctivitis, in which event some other mydriatic must be substituted. Homatropin and scopolamin are suitable.

Inflammation of the conjunctiva of the inner canthus is a frequent complication of dacryocystitis. Relief usually attends restoration of tear drainage.

INJURIES OF THE CONJUNCTIVA AND THEIR EFFECTS

Burns of the conjunctiva may occur from molten metal or chemicals. Ulceration is to be expected, and the chief concern is to obtain healing without contraction of the membrane, and without symblepharon. Infection is to be avoided by antiseptic treatment. Ulcers must be kept clean. In the stage of healing, connective tissue is proliferated in superabundance.

Contractions are apt to form regardless of the kind of treatment, and entropion with trichiasis is probable. Union of the palpebral and ocular conjunctivæ is prone to occur. The membranes may be kept separated during the healing process by inserting oiled pledgets of cotton deeply into the wound between them; this will encourage healing below. When entropion or trichiasis begins, one of the entropion sutures is indicated. After healing is complete, a plastic operation may be required.

Symblepharon (see Fig. 4), union of the bulbar and palpebral conjunctivæ, requires operative correction when it is sufficiently extensive to deform the lids, produce disfigurement, impede the rotations of the globe with resulting diplopia, cause irritation, involve the cornea or prevent complete closure of the lids. The amount of correction needed will depend upon the condition present. When the union is narrow, the membranous band is to be dissected entire from the globe as far as the fornix.

With a double-armed suture the dissected portion is drawn as deeply into the fornix as possible, and the needles are passed out to the skin surface near

the margin of the orbit. When entropion has formed, a Snellen's suture can be completed. The denuded scleral surface is covered by undermining the conjunctiva in each direction from the wound, and uniting the edges with sutures.

When the adhesion is broad, Teale's operation is to be considered. The membrane is dissected from the globe as before, and drawn into the fornix. Pedicled flaps are cut from each side of the cornea, rotated to cover the denuded surface of the sclera, and sutured in place. The edges of the conjunctiva are united over the sites from which the flaps were cut.

Where the ocular conjunctiva is insufficient to supply a graft for an extensive symblepharon, a flap may be fashioned from the folds of the superior fornix. The lid is dissected from the globe; the flap is fitted into place and secured with fine silk sutures. Both eyes should be bandaged to prevent any movement of the globes for four days or more. Then the dressings are removed and the secretions are wiped away. Dressings should be continued until firm union of the graft has taken place. Transplants of rabbit conjunctiva have been disappointing. The adhesion may affect the membrane near the border of the lid only, and not extend into the fornix; it can be divided. The parts are kept separated until each surface acquires epithelium.

In wounds of the conjunctiva, it is important to thoroughly inspect the entire membrane for possible injury. Lacerations are repaired by uniting their edges, and then guarding against symblepharon.

NEW GROWTHS ON THE CONJUNCTIVA

Pinguecula is a small rounded elevation of yellowish color, situated near the limbus in the palpebral fissure, usually at the nasal side; it is harmless. It must not be confused with the beginning of a pterygium. For cosmetic reasons it may be excised.

Pterygium is a triangular fleshlike thickening or hypertrophy of the conjunctiva (Fig. 87). It usually grows from the medial fornix and converges toward the cornea, or upon it, in a head which is rounded and elevated. A true pterygium is not united to the globe at the corneoscleral junction; a small probe can be passed beneath it. When it is permitted to grow over the cornea, it will in time cover the pupillary area to the detriment of vision. Pterygia always occur in the palpebral fissure, and grow on either side of the cornea. They are disfiguring, they may restrict the movements of the eyeball, and they injure sight. Operative treatment is indicated.

The conjunctiva is anesthetized with cocain, and the conjunctival sac is cleansed with 1 : 8,000 solution of mercuric chlorid. The lids are separated

with a speculum. The pterygium is grasped at the limbus with tissue forceps and lifted up. With a small sharp scalpel the head is dissected smoothly from the cornea. With small straight scissors the growth is separated from healthy conjunctiva along its lower border. Beginning at this incision the conjunctiva is undermined below the cornea to form a pocket. From the corneal end of the incision the conjunctiva is divided upward for a few millimeters. The pterygium is then undermined.

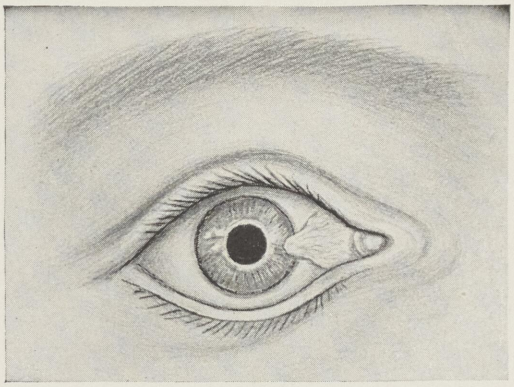


Fig. 87.—Pterygium.

Both needles of a double-armed silk suture are passed through the head of the pterygium from its under surface, and down into the pocket. They are brought out to the surface of the conjunctiva near the fornix 3 or 4 millimeters apart (Fig. 88); the head of the pterygium is then drawn deeply into the pocket. The suture is tied (Fig. 89). The upper border of the pocket can be sutured to healthy conjunctiva from above the transplanted pterygium. No sutures will be required for the vertical incision next the cornea.

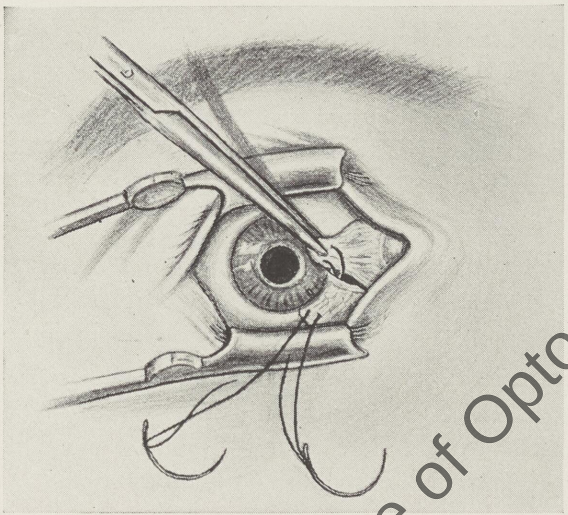


Fig. 88.—McReynolds' Operation.

The corneal surface from which the growth was removed should be curetted and shaved until it is smooth and free of shreds. White's ointment is applied and a pad is adjusted. The dressings are omitted after two days, and the suture is removed on the fifth day. This is essentially the operation devised by McReynolds. It is most useful, because the pterygium can continue to grow and yet do no harm. The transplantation can be made above if necessary.

Arlt's operation is performed as follows: Dissect the head from the cornea as before. From the upper and lower points where the pterygium crosses the margin of the cornea, converging incisions are made back through the body of the growth. This flap is removed from the sclera by dissection, and the de-

nuded area is diamond shaped (Fig. 90). Short vertical incisions are made up and down alongside the cornea. The incised edges of the pterygium are then approximated with interrupted sutures. Relapses are not uncommon.

Some operators strip the head from the cornea with a muscle hook which has been inserted beneath the unattached neck. Others pass a thread under the neck and saw the head free from the cornea.

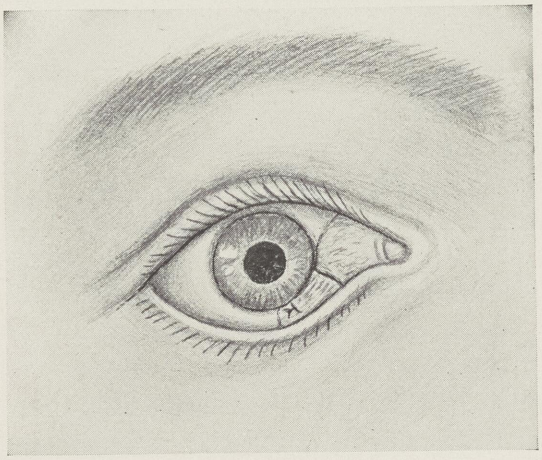


Fig. 89.

Decapitation of the pterygium is never advised. When the patient refuses a classical operation, or if for any reason it cannot be done, decapitation can be accomplished with the galvanocautery, or the circulation can be interrupted by tying a silk thread tightly around the neck.

In some situations a pterygium may resemble a pannus.

Pseudopterygia form from diseases or injuries of the conjunctiva and cornea. A probe cannot be passed beneath them at the limbus. A simple excision with closure of the conjunctival wound usually suffices. It is more like a symblepharon than a pterygium. It is nonprogressive. Sometimes simple division of the neck at the limbus allows the growth to retract and atrophy.

Ecchymosis or subconjunctival hemorrhage is denoted by a bright uniform red area without vessel network. It often occurs some hours after a skull injury, in which the blood extravasates forward to the conjunctiva. It may follow straining, as in whooping-cough. Recurrences suggest arteriosclerosis, chronic nephritis and diabetes. Usually no treatment is effective. Tense swelling may require a pressure bandage, and excessive turgescence may demand scarification of the conjunctiva.

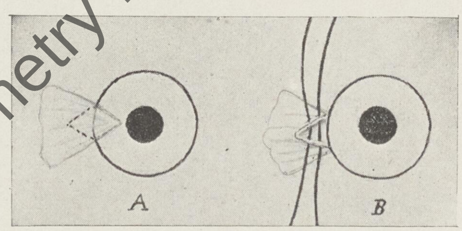


Fig. 90.—Arlt's Operation. (After Meller.)

Edema or chemosis of the conjunctiva is a sign of disease or injury. When emphysema occurs, injury of the ethmoidal bone is suspected.

Concretions often work their way toward the surface of the conjunctiva. They give the same sensation as the presence of a foreign body, and as a rule

manifest themselves abruptly. They are readily found as small white or yellowish points, and are to be removed, after cocainization, with a needle or sharply pointed knife. White's ointment is applied, but no dressing is needed.

Tumors and cysts of great variety, although rare, may grow in the conjunctiva.

CHAPTER V

THE CORNEA

ESSENTIALS OF THE ANATOMY

The outer coat of the eyeball is composed of the cornea and sclera. The anterior sixth of the circumference is cornea, and the remainder is sclera. The cornea consists of five principal layers: The anterior epithelium, Bowman's membrane, the substantia propria, Descemet's membrane and the endothelium (see Fig. 103).

The epithelium is composed of cells arranged in from five to seven layers. The deepest cells are cylindrical; the succeeding layers are more and more cuboidal and flattened, and the most superficial are of pavement type. Because of the constant presence of moisture the superficial cells do not keratinize, become opaque, and desquamate like the devitalized cells of the skin, but are swept up by the lid margins and washed away by the tears. Bowman's membrane, or the anterior elastic membrane, is a homogeneous structure.

The substantia propria is built up of flattened bundles of fine fibrous tissue. The bundles are arranged in laminæ; the fibers in one bundle form broad angles with those in others. The spaces between the bundles are occupied by fixed and motile corneal corpuscles. The spaces are connected by canals, so that fluid may flow from one part to another.

Descemet's membrane, or the posterior elastic membrane, is also a homogeneous structure. The endothelium consists of a single layer of flattened cuboidal cells which excludes the aqueous humor from the substantia propria. It is continued over the fibers of the pectinate ligament and over the anterior surface of the iris, but does not enter the iris crypts.

The cornea is oval; its horizontal diameter is 12 millimeters, and its vertical diameter is 11 millimeters. The sclera overlaps the cornea more from above and below than it does from either side. The radius of curvature of the anterior surface is about 7.8 millimeters, while that of the posterior surface is about 7.5 millimeters. The thickness varies from 0.9 millimeter in the center to 1.1 millimeter at the limbus.

Clinically the cornea may be considered in three layers. The most superficial includes the epithelium, Bowman's membrane and some anterior bundles of the substantia propria; they are continuous with the conjunctiva and the

episclera and are subject to the same diseases. The deepest layer includes the endothelium, Descemet's membrane and a few posterior bundles of the substantia propria; these are continuous with the iris and ciliary body and react to the same diseases. The greater part of the substantia propria is continuous with the sclera.

The cornea has no blood-vessels; from the arterial loops, described under the blood supply of the bulbar conjunctiva, nutritive materials circulate through the corneal tissues. The nerves of the cornea are sensory, from the nasociliary branch of the ophthalmic, through the ciliary nerves, and directly from the ciliary plexus. Superficial branches supply the outer layers of the cornea, and deep ones supply the central part.

The nerve bundles break up into fine fibrils which pass through pores in Bowman's membrane. The fibrils terminate in the epithelium by end-bulbs and fine endings. These terminals are exquisitely sensitive. Their connections establish a balance in the secretion of tears, and the frequency of winking to provide for cleansing and polishing the corneal surface; this is essential to uniform vision. The direct sensory effect gives immediate information of the presence of any foreign substance on the cornea, and usually, not always, of injury to the cornea.

EXAMINATION

Examination of the cornea is accomplished by a variety of methods. The patient is seated facing a light, the lids are separated, and the eyes are inspected and compared. The patient is requested to look in various directions, which brings into view successive portions of the cornea and conjunctiva of each globe.

It is important to note any engorgement of the fine vessels around the cornea. Pannus and vascular invasions direct attention to trachoma, keratitis, ulcers and healing wounds. The size and shape of the cornea, and the smoothness, polish and luster of its surface are noted.

The cornea should be transparent throughout, so that the whole iris pattern is uniformly visible. Where a portion of the iris surface is obscured, it may be due to a change in the iris itself, to a localized turbidity in the aqueous humor, or to an opacity in the cornea. The eye is slowly rotated in various directions; if the spot moves with relation to the pupil as a fixed point, iris change is excluded. The iris pattern may not be clear in uveitis and glaucoma; in either of these conditions the cornea may lose transparency. It should be noted if the anterior chambers are equally deep.

The smoothness of the surface can be tested with the shaft of a hatpin. As the wire is moved before the cornea in various directions its image should be

uniformly straight and continuous. Unevenness will be detected by distortion of the image. The state of polish may be determined by the sharpness or dullness of the image, particularly when compared to a normal cornea.

Placido's disk is a flat plate with alternating rings of black and white on its face (Fig. 91). A tube with a plus 4 lens is fitted to a hole in the center of the plate. The patient is seated facing from the light, and in such a position that the disk is illuminated. The surgeon looks through the tube at the eye from a distance of 25 centimeters (10 inches), and the patient looks at the hole in the plate. The white rings are mirrored in the patient's cornea, and the examiner observes the regularity of curvature and uniformity of width of the images.

Fig. 91.

The sensitiveness of the cornea is tested by touching the surface with the end of a wisp of tightly twisted clean cotton. Both should be tested for comparison. The reaction is involuntary winking. The lid margins, lashes or conjunctiva must not be touched. The cotton must not come into the pupillary area, where a visual winking reflex might be stimulated.

Photophobia, increased lacrimation, pain and blepharospasm accompany many injuries of the cornea and some of its diseases. These patients, frightened children, and timid individuals require special preparation for examination. Cocain or phenacain in 4 per cent solution, or butyn in 2 per cent solution, is instilled in both eyes. The instillation is repeated. The room light should be reduced during the preparation, and perhaps during the examination. When the blepharospasm relaxes, the lids are separated with the surgeon's fingers or with retractors or muscle hooks.

Fig. 92.

Force must not be used in these cases; patience, gentleness and inspired confidence will usually accomplish the desired end. When persuasion fails, an anesthetic must be given. The condition of the cornea must be known.

Oblique illumination is practiced by seating the patient in a darkened room with a good light in front of and to one side of the face. A condensing lens of 13 to 16 diopters is used to focus the light on the eye. The light is focused on the aqueous chamber and the iris in turn. Moving the lens nearer to or farther from the eye will permit examination at various depths (Fig. 92). By moving the point of focused light, or by having the patient move his eye, the entire area and thickness of the cornea can be explored.

The surgeon uses his unaided vision, or a magnifying glass or a loupe. The largest magnification is obtained when the surgeon brings his eye as near

the magnifying glass as he can without blurring the image of the light on the cornea. A binocular loupe is used like a magnifying glass.

The ophthalmoscope also is used for examining the cornea. When an opacity is illuminated it reflects the light and shows its color. When the light is reflected back by the fundus, an opacity always appears black because it obstructs the reflected rays (Figs. 93 and 94). With a plus 7 lens in the sight hole of the instrument, the light is focused on the iris by varying the distance between the observing and observed eyes. The patient slowly rotates his eye in various directions while the examiner looks at the pupil.

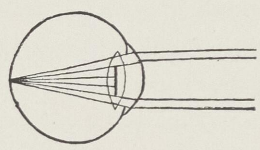


Fig. 93.—Opacity in Lens.

The presence of an opacity is detected in the illuminated area of the pupil. When it moves in the same direction that the eye moves, it is in front of the pupil and probably in the cornea. When the opacity is relatively stationary, it is in or near the plane of the pupil; when it appears to move opposite to the direction that the eye moves, it is behind the pupil (Fig. 95).

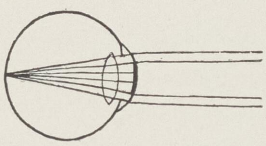


Fig. 94.—Opacity in Cornea.

Instead of having the patient move his eye, he may be directed to gaze at a fixed object while the examiner shifts his own position in various directions to study the apparent movement of an opacity; by this method the opacity appears to move in a direction opposite to the movements described when the eye is rotated.

With a plus 16 or plus 20 lens in the sight hole of the ophthalmoscope an opacity can be illuminated and examined by direct magnification at a distance of 5 to 7 centimeters (2 to 3 inches).

The cornea may be examined by staining; any part that is deprived of epithelium will stain. The method is of value in erosions, abrasions, penetrating wounds and ulcers. Mercurochrome 220 in 2 per cent solution produces a red stain, but does not afford the contrast offered by fluorescein 2 per cent combined with sodium carbonate, not bicarbonate, 4 per cent solution.

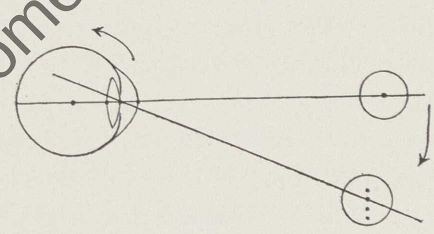


Fig. 95.

The cornea is anesthetized; the lids are separated, the patient is told to look downward, and a drop or two of the stain is floated over the cornea. The lids are closed. After a few minutes the eye is irrigated with boric acid or normal salt solution. A simple loss of epithelium is denoted by a bright green; necrotic tissue stains yellowish.

Where the lesion extends as deeply as Descemet's membrane the latter does

not stain, but the surrounding injured tissue is colored; the stained area has a dark center. Descemet's membrane is elastic and bulges up into a wound. This is a descemetocele. When touched with a probe it dimples without affecting surrounding tissue. A foreign body embedded in the anterior corneal layers does not stain, although surrounded by stained tissue. When it is touched contiguous tissues dimple also. Careful scrutiny will usually indicate the depth of staining. The stain is used also to verify the removal of a foreign body.

The preliminary use of cocain intensifies the action of fluorescein, but after repeated instillations it loosens the epithelium, and the cornea may take on a dull diffuse stain. Fluorescein will color the endothelium in some of the inflammatory uveal diseases, while the epithelium remains sound. This stain is faint and diffuse.

Eosin or methylene-blue may be employed as stains.

INJURIES OF THE CORNEA

The general practitioner is called upon to care for many cases of foreign body "in the eye." The usual symptoms are pain of sudden onset, increased lacrimation, and blepharospasm. Photophobia is seldom present at first.

Particles of coal, cinder, emery, metal, stone and glass constitute the bulk of foreign bodies that injure the eye. To gain some idea of the material before attempting its removal, the patient should be asked what he was doing or where he was at the time of injury. The occupation is suggestive. The physician will prepare a cotton-wound probe or toothpick and have it within easy reach.

The skin of the upper lid is grasped between the finger and thumb and the lid is lifted from the globe; this ordinarily affords relief, but the pain returns when the lid is released. If this does not relieve, the lower lid is rotated from the globe and the conjunctiva is searched. The upper lid is then everted. The palpebral conjunctiva is inspected, then the bulbar, the cornea, and lastly, the upper fornix. If the foreign body is found, an immediate attempt can be made to remove it with the prepared probe. If the body is removed and the patient is comfortable, one may be assured of relief, except for infection, for up to this time no anesthetic has been used.

If the foreign particle cannot be located because of blepharospasm, nor removed because of sensitiveness or pain, cocain, butyn or phenacain is indicated.

Should the blepharospasm be uncontrollable, 16 milligrams ($\frac{1}{4}$ grain) of morphin sulphate in 1 cubic centimeter ($15\frac{1}{2}$ minims) of 2 per cent novocain solution can be injected into the temporal area of the affected side. This

makes the patient comfortable, and a thorough search can be made for the foreign body.

Embedded foreign bodies have to be lifted out of the tissues with the point of a sharp paracentesis needle or a spud (Figs. 96 and 97) designed for that purpose. The surgeon wears a loupe and focuses light on the body with a convex lens; the patient is directed to fix his gaze on a definite point and keep his attention on it. The lids are separated with a speculum or the fingers of the hand that holds the condensing lens. By shifting the position of the light, the lens, the patient's direction of gaze and the examiner's angle of vision, the body can be illuminated and magnified so that its removal may be accomplished with safety.

Fig. 96.—Needle.

Magnetizable metal can be extracted with an eye magnet. Where a splinter of metal extends through the cornea, it may be necessary to insert a keratome beneath it to supply a surface against which to work, or with which to push it back through the cornea until it can be grasped with fine forceps.

Iron particles usually leave rust which is visible after extracting the metal; it can be removed with a dental drill rotated by the fingers. Jackson has suggested that powder grains be touched with a fine-pointed galvanocautery needle. Particles of glass are difficult to find. A wound stained with fluorescein furnishes a guide to exploration with the point of a knife; contact with a hard body imparts a tactile sensation to that effect. Many kinds of glass are not opaque to x-rays.

Fig. 97.—Spud.

The movements of the lids and globe generally lacerate the tissues against the foreign body and delay healing; the patient returns and says that the foreign material was not all removed. The suggested use of novocain to paralyze the motor nerves to the lids is useful in this connection. It prevents the lids from pressing on the wounded surface, and favors healing by the absence of such pressure.

After treatment for foreign body the conjunctival sac is irrigated, one drop of 1 per cent atropin sulphate solution is instilled, White's ointment is placed beneath the lids, and a pad is worn for twenty-four hours. Infection may arise; the patient is advised to report promptly if the eye becomes uncomfortable.

A foreign body should be removed promptly; otherwise it is usually covered with epithelium and forms a small abscess. This ruptures in time and the foreign body is expelled. Unless epithelial cells fill it (see Fig. 103) a permanent depression remains. The abscess may heal without rupturing and interfere with vision, or fail to heal and destroy the globe.

Perforating Injuries of the cornea sometimes include the lens or the iris. If the latter, blood will probably appear in the anterior chamber. The conjunctival sac is cleansed with 1:5,000 bichlorid of mercury solution, and a flap of conjunctiva is brought over the wound (Fig. 98). If the latter is central it may be necessary to undermine the conjunctiva around the cornea and draw it up with a purse-string suture of five-day catgut.

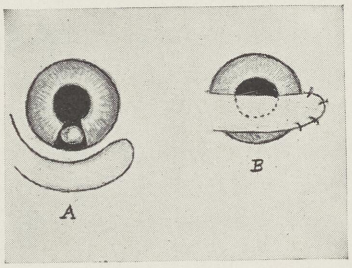


Fig. 98.
(After Meller.)

In appropriate cases other conjunctival flaps may be fashioned (Fig. 99). Any half of the cornea may be covered by separating the conjunctiva from the corneal margin for a suitable distance; the membrane is then undermined sufficiently to be drawn over the wound where it is fixed in position by sutures (Fig. 100).

The sutures are less likely to tear out if they are placed by cross bights. The needle enters the conjunctival flap 8 or 10 millimeters from the limbus, and is directed toward the cornea. It is brought out 2 or 3 millimeters from the point of entrance. The needle then enters the undisturbed conjunctiva, is directed away from the cornea, and is brought out 2 or 3 millimeters distant. One such suture is placed on each side of the cornea, and the flap is drawn evenly over the wound.


Fig. 99.

Before the sutures are tied, shreds of tissue are trimmed away from the wound, and small prolapses of iris are cut away. Atropin is then instilled, White's ointment is applied to the conjunctival sac and the eye is dressed with a pressure pad. The sutures are removed after five to seven days. Injuries limited to the cornea heal readily in the absence of infection. Infection may be brought to the eye by the injuring body, or it can come from dacryocystitis. If the cornea is injured and the lacrimal sac is under suspicion, the puncta should be sealed with the galvanocautery.

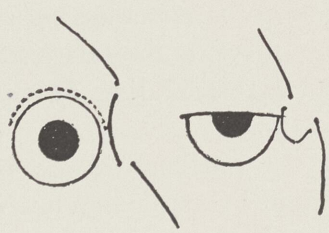


Fig. 100.

Burns of the cornea occur from scalds or flame, from hot metal in some occupations or from contact with a curling iron, and from acids, alkalies, or unslaked lime. The depth of a burn may vary from a superficial destruction of the epithelium to the entire thickness of the cornea. A burn extending through the cornea is treated like a perforating injury.

Antiseptic cleansing, White's ointment, atropin, and a pad dressing are

indicated for all burns that do not perforate. Where pain is severe, the motor nerves of the lids should be infiltrated with novocain and morphin solution. Dressings are made daily until healing is completed. Atropin is indicated except in cases where the intra-ocular tension is raised, or where it is desired to draw the iris away from a wound near the periphery of the cornea; in such instances eserin is employed in 0.25 per cent solution, one drop once or more daily.

Splashes of hot metal may strike the eyeball and settle into the conjunctival sac; the particles must be removed.

Acid burns should be irrigated promptly with a cold solution of bicarbonate of soda, and alkali burns with weak vinegar or diluted acetic acid, although the surgeon will seldom see a case in time to effectively neutralize acid. Careful search must be made for particles of alkali and shreds of tissue; these are to be removed with forceps or with cotton wound on a probe. The treatment is the same as for heat burns.

Lime burns are irrigated like alkali burns. Sugar water will precipitate the lime, but the chemical combination generates heat. If the cornea can be anesthetized, the irrigation may be done with a freshly prepared 2 per cent solution of ammonium chlorid or neutral ammonium tartrate. As improvement occurs the strength can be gradually increased up to 20 per cent. Patience is required to free the cornea of all lime particles. They tend to imbed in the cornea, undergo chemical modification and produce dense opacities. Dionin in 1 to 5 per cent solution allays pain and is said to favorably modify the tendency to the formation of corneal opacities due to lime.

Chemical burns nearly always involve the conjunctiva, and especial attention must be given to cleanse it thoroughly and to prevent symblepharon.

In all painful burns the application of cold is more soothing than heat. Cold compresses, phenacain ointment, and immobilization of the lids with novocain and dressings will obviate the necessity for morphin in many cases.

Abrasions and Erosions of traumatic origin refer to losses of epithelium. The lesion can be verified by fluorescein staining. The injuries are caused by foreign bodies, leaves, twigs, wheat beards, and so forth.

Infants sometimes injure their mothers' eyes with their fingers nails. This wound is exquisitely painful, as the epithelial cells seem to be stripped off the sensory nerve-endings, leaving them exposed (Fig. 101). The treatment for heat burns is applicable. The lids must be paralyzed with novocain and morphin to abolish the intense blepharospasm.

When the pain of epithelial injuries is contrasted with the lesser pain that attends many gouged-out wounds that extend into the substantia propria, it seems that in the latter form the nerve-endings are broken off (Fig. 102-A).

Erosions of the cornea may apparently heal, but the epithelium is not firmly adherent to Bowman's membrane. After some days, weeks or months the eye becomes painful. The discomfort is particularly noticed on awaking, but passes off after a few hours. The pain returns for one or a few mornings, and then disappears. Later on it recurs. The mechanism is explained as follows: The lids adhere during sleep; upon awaking they are forcibly separated and the new epithelium is torn off (Fig. 102-B). The covering is restored in a few hours and the pain subsides. Whenever the lids adhere so that they have to be forcibly opened, the process is repeated.


Fig. 101.

The objects of treatment are to gain firm union between the epithelium and Bowman's membrane, and to prevent the lids from adhering together. The latter can be accomplished by the use of an ointment and a bandage at bedtime. The former requires some form of mild irritation, and dionin in 1 to 3 per cent ointment meets both indications. Phenacain, 1 per cent, may be added when the eye is irritable. This ointment is used at night for one week. On alternate weeks one drop of sterile castor oil is used; the bandage is retained. Yellow oxid of mercury 1 or 2 per cent ointment is useful also.

Recurrent erosions of the cornea sometimes occur independent of any known injury. The onset, symptoms and management are the same. These cases require treatment over an extended period of time, or until several months have elapsed since the last attack.

These erosions possess but faint staining qualities, and fluorescein produces a pale gray-green instead of the characteristic bright green stain. In persistent intractable cases some surgeons scrape the epithelium from the affected area, and allow it to regenerate from the base. Others cover the cornea with a conjunctival flap, and bandage the eye for two weeks.

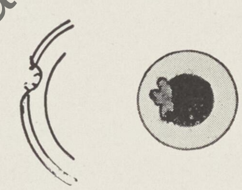


Fig. 102 A and B.

Contusions or bruises of the cornea are followed by an infiltration or an edema of the tissue which somewhat resembles interstitial keratitis. The history of injury and the early recovery will differentiate the conditions. When pain is present, hot or cold applications, and an ointment of 1 per cent phenacain with 1 to 5 per cent dionin will likely control it. Massage of the cornea often hastens recovery. In cases of congenital syphilis, a wound of the cornea may incite a true interstitial keratitis.

Rupture of the cornea without rupture of the sclera rarely occurs; it is to be treated as a penetrating wound of the cornea. Where the tear is near the corneal margin the iris will probably prolapse; care must be exercised in excising such a protrusion, for the ciliary body may lie immediately behind or be

included with the prolapse. Where the latter is large, an attempt should be made to replace it; eserin 0.25 per cent solution is instilled to contract the pupil and hold the iris out of the wound. Suture of the cornea may be undertaken, but a conjunctival flap snugly applied to the rupture will probably prove satisfactory.

Bowman's membrane is often destroyed at the point of a corneal injury. It is claimed that this membrane is never regenerated (Fig. 103). In such cases the epithelium grows into the wound and rests upon the substantia propria or upon newly formed connective tissue. Where the wound is healed by a proliferation of corneal cells, the cornea will regain transparency. A permanent opacity usually marks the sight where a proliferation of connective tissue fills the wound.

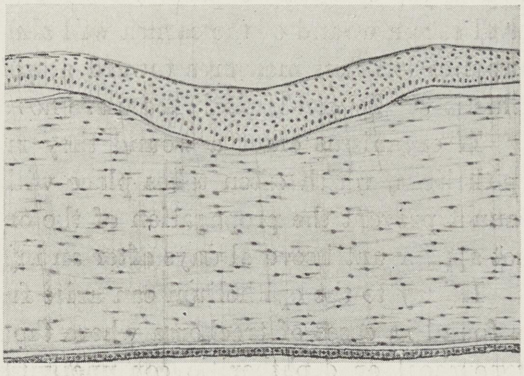


Fig. 103.—Healed Cornea, Destruction of Bowman's Membrane. (After Fuchs.)

Where the epithelium fails to regenerate to the former level, a facet remains. Such facets are occasionally found after the healing of ulcers caused by measles, and these facets are frequently found in the pupillary area.

Blood Staining of the cornea may follow blows on the eye, particularly when the anterior chamber has filled with blood. This is especially prone to occur when the intra-ocular tension is raised. The color ranges from red to brown or green. When it is green it must be differentiated from dislocation of the crystalline lens into the anterior chamber (Parsons). The coloring matter of the blood, and not corpuscles, has been found in the cornea. Clearing begins from the periphery, and a long time elapses before transparency is restored. Bilateral blood staining is very rare.

INFECTIONS AND VESICULATION

Every foreign body is a potential carrier of infecting organisms. A wound of the cornea may become infected by the introduction of organisms from without, exogenous infection, or from within, endogenous infection. The latter is provided by foci in the teeth, tonsils or elsewhere in the body. The process is facilitated by the local hyperemia that initiates healing.

The kind of organism, its virulence and the depth of its invasion into the

corneal tissues largely determine the character of the resulting lesion. Keratitis, ulcers and abscesses may occur; any of these can heal with loss or restoration of tissue, and with loss, diminution or recovery of vision.

The flora of the conjunctival sac has no influence on an intact cornea, but when a breach occurs in the epithelium the same flora becomes active. The most potent organisms are pneumococci, streptococci, staphylococci and diplobacilli. A diseased lacrimal sac usually contains pneumococci; the act of wiping the eyes may express the contents of the lacrimal sac into the conjunctival sac; a wound of the cornea will almost certainly become infected. Nature supplies the first measures toward asepsis by irrigating the wound with tears. This is wholly mechanical; tears are not antiseptic.

If organisms enter a wound they may be sealed in by the restoration of epithelium, which often takes place within twenty-four hours. This covering cannot prevent the propagation of the organisms. An ulcer or an abscess may not appear until several days after an injury.

Injury to the epithelium can arise from the irritation due to trichiasis. It is found in cases of trachoma where the rough lid scratches the cornea, and in gonorrheal or diphtheritic conjunctivitis where the epithelium is macerated by the secretions. Drying of the epithelium occurs from infrequent winking, inability to close the lids, malnutrition or keratomalacia, exhausting diseases, and after prolonged use of cocain; such epithelium is unable to protect underlying tissues. Accidental exogenous infection occurs readily without trauma in a cornea whose epithelium has been disturbed by vesicular eruptions.

Endogenous infections frequently occur after the eyeball has been opened surgically, either coincidentally with the healing process or after healing is apparently complete. These infections are usually situated deeply within the cornea. They are accompanied by some degree of edema in all the layers, presumably from an excess of fluid in the intercellular spaces. This probably accounts for the deep gray-green fluorescein stain which is sometimes seen when the epithelium is apparently intact.

The behavior of new blood-vessel formations is more comprehensible when the anatomy of the blood supply is reviewed. The circumcorneal loops are in the episcleral tissue surrounding the cornea; in them the blood column is plainly visible under low magnification. These and the conjunctival vessels become engorged in superficial inflammations of the cornea.

The deeper tissues next the limbus are supplied principally by branches of the anterior ciliary arteries. Being deeply covered, the dusky blood columns are ordinarily visible only in deep-seated inflammations. Each system, superficial and deep, has its peculiar manifestations in the extension of vessels into the diseased area. Both systems may be engorged at once.

New vessels from the conjunctiva and the circumcorneal loops are easily seen in the cornea by reason of their brighter color and because they slightly elevate the epithelium. These branch freely, and often extend to the center of the cornea (Figs. 104 and 105). The deeper vessels are straight; they do not branch, or extend so far into the cornea. They are difficult to see, and may be invisible if the cornea is clouded. These vessels are more highly engorged when the iris and ciliary body are congested (Fig. 106).

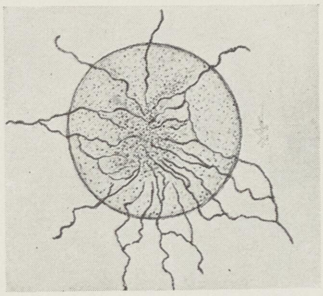


Fig. 104.—Pannus. (After Fuchs.)

The purpose of the vascular invasion is to combat infection and repair the damage wrought by it. The infection spreads more rapidly than the blood-vessels can grow; they follow the infection but rarely overtake it. Connective tissue is proliferated, and this accounts for the permanent opacities in the paths of the lesions.

A cornea loses its luster and is clouded in an area corresponding to the site of the infection. The luster is lost because edema produces a roughness of the surface. The clouding is due to an infiltration with polymorphonuclear leukocytes and perhaps to some proliferation of corneal cells. The density of the infiltrate determines the degree of the clouding. The severity of the infection and the energy of defense are indicated by the degree of engorgement of the circumcorneal vessels. When the response is prompt and vigorous the prognosis is more favorable.

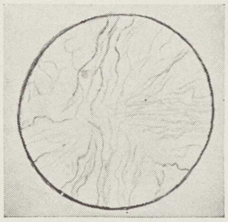


Fig. 105. (After Fuchs.)

Where the iris and ciliary body are congested, leukocytes escape from the blood-vessels and accumulate in the anterior chamber or against the endothelium of the cornea. These cells for the most part remain free in the aqueous and gravitate to the lowest part of the anterior chamber; this is called hypopyon (Fig. 107). It disappears as the disease is brought under control. Occasionally a fibrinous exudate is poured out with the cells and holds them in suspension; in such cases the anterior chamber does not so promptly become clear.

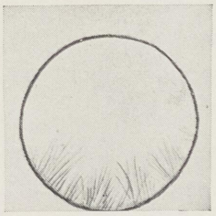


Fig. 106. (After Fuchs.)

Very few of the infections under consideration attack children, although interstitial and phlyctenular keratitis are notable exceptions. In older people the nutrition of the cornea is impaired, so that repair after injury is delayed and infection is more prone to occur. Because of poor nutrition also, corneal diseases are found more frequently among the poorer classes. More cases of these disorders are found in dispensary than

in private practice. It seems also that aliens are oftener infected than are native Americans. Strictly agricultural communities show a relatively small percentage of eye diseases.

Vesiculation of the Cornea occurs from various causes. As the epithelial layer is involved, some of the symptoms of a foreign body are usually present; these are increased lacrimation, blepharospasm, pain and photophobia. Vision is often impaired. Multiple staining areas and the absence of a history of injury facilitate diagnosis. Sometimes the cornea is hazy but does not stain. The haze is usually due to multiple minute opacities beneath the epithelium.

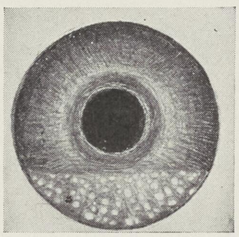


Fig. 107.

While vesiculation ordinarily appears in febrile states, it may accompany endogenous infections such as iridocyclitis. In both instances, staining will occur at some stage of the disease, but it is atypical, as in recurrent erosions. Although pain is present, the sensibility of the cornea is usually diminished.

The vesicles are covered lightly and rupture readily with ordinary lid movements. The vesicle may break on one side and leave a filament or shred of tissue hanging from the cornea; this is called *filamentary keratitis* (Fig. 108).

Fig. 108

Herpes Febrilis of the cornea is due to the same diseases that produce herpes facialis and herpes labialis. While the eruption may appear only upon the cornea, it generally follows the appearance of similar lesions on the face or lips. As the vesicles sometimes cause opacities, they require special treatment. Atropin, phenacain ointment and a protective bandage are indicated.

Herpes Zoster of the cornea is a variation of herpes zoster ophthalmicus (*q.v.*). Corneal sensation is diminished. The course is prolonged and permanent opacities may form. The symptoms are severe, and iritis is a frequent complication. The local treatment is the same as for febrile herpes; dionin 1 to 5 per cent may have to be added to the ointment to control the pain. The prognosis for vision may be unfavorable because of permanent opacities.

KERATITIS, SUPERFICIAL FORMS

Dendritic Keratitis may be associated with malaria, although it exists when malaria cannot be demonstrated. It is a branching form of superficial corneal ulceration, and one or several branches may be present (Fig. 109-A). Symptoms may be absent, or they may be severe and produce the sensation of a rough foreign body. The lesions stain readily, and in some cases the entire cornea

accepts the stain. The branching characteristic is practically diagnostic. The disease is of long duration and recurrences are common. Sometimes vision is permanently impaired.

Treatment.—Pure phenol is applied to the lesion. A sharply pointed wooden stick is dipped into the pure acid and allowed to absorb some of it. No excess must be present on the wood. The cornea is anesthetized with phenacain or butyn, the lids are held apart until the cornea is slightly dry, and then, with the aid of a magnifying glass or loupe the carbolized point of wood is applied to every part of the ulcer.


Fig. 109-A.
(After Parsons.)

The phenol is followed by atropin, White's ointment and an eye pad. If a complicating infection occurs, the process is inclined to spread over the whole cornea. The prognosis is unfavorable when complications exist, but is fairly favorable in uncomplicated cases of short duration.

Phlyctenular Keratitis is properly considered in connection with phlyctenular conjunctivitis (*q.v.*). It occurs in superficial and deep forms; the former is more common. The lesions form at or near the limbus (see Fig. 85). They are discrete and often multiple; they frequently develop, rupture, and heal without spreading. If they form all around the margin of the cornea they constitute a ring abscess and can destroy the eyeball.


Fig. 109-B.
(After Nettleship.)

A phlyctenule may originate near the limbus and creep toward the center of the cornea, but it rarely reaches the pupillary area. This is one form of serpiginous ulcer. Newly formed vessels spring from the corneal loops, follow in the path of the ulcer, and promote healing as the lesion advances; this is vascular or fascicular keratitis (Fig. 109-B). The lesion is superficial and destroys epithelium. The latter does not fully form over the healed tract, and a shallow groove remains. When healing is completed, the new blood-vessels largely atrophy. A permanent opacity marks the path of the ulcer.

The deep variety forms beneath Bowman's membrane. These lesions infiltrate or spread beneath the surface; they are gray at first, but become yellowish. This denotes abscess formation. They may not only rupture on the surface, but may perforate into the anterior chamber. Blood-vessels grow from any part of the limbus toward the lesion and form a pannus.

Phlyctenular keratitis affects children especially and occurs in crops. Photophobia, lacrimation, blepharospasm and pain are severe. The child insists on staying in a dark room with its face buried in a pillow, although the relief is more imaginary than real. The irritation affects the corneal nerves directly, and light only serves to aggravate the pain. Therefore protection from light, other than by dark glasses, is not advised.

Treatment consists of correcting refractive errors, regulating the diet and prohibiting sweets, pastries and fried foods, attending to the general physical condition, hygiene and habits of living, directing outdoor exercises, and prescribing cod-liver oil and syrup of iodid of iron as indicated.

Local treatment serves to arrest the development and progress of the lesions, and thereby protects vision. Ointments of 1 or 2 per cent yellow oxid of mercury are indicated. Calomel in fine powder may be dusted on the cornea with a camel's-hair brush. Phenacain 1 per cent ointment is soothing and beneficial. When the lesions are indolent, two drops of 1 or 2 per cent solution of silver nitrate should be instilled, and neutralized with normal salt solution.

Ulcers should be touched with pure phenol as for dendritic keratitis. When it is desired to destroy the pannus, it is best done with the galvanocautery. It will be necessary to first anesthetize the cornea and conjunctiva with butyn, phenacain or cocain.

Irritation of the iris, ciliary body or the deep layers of the cornea is indicated by engorgement of the fine violet-colored deep vessels at one or more points surrounding the cornea. The pupil should be kept dilated with atropin.

Acne Rosacea is sometimes accompanied by an acne keratitis which resembles phlyctenular keratitis. The presence of acne on the face will give a clew; the acne must be treated or the corneal condition will not heal. This disease affects adults. Calomel is dusted on the cornea from a camel's-hair brush.

Marginal Keratitis occurs in elderly people; it forms about the margin of the cornea. The lesions are shallow ulcers of a persistently recurring nature. They may encircle the cornea, break down and form ring ulcer. This tends toward destruction of the cornea. The local treatment is the same as for phlyctenular keratitis.

Superficial Marginal Keratitis occurs in people of middle life; the lesions are shallow. It has the characteristics of a mild ulcer, and tends to involve successive portions of the corneal margin. The lesions creep toward the center of the cornea but do not reach it. A faint opacity remains, but as the pupillary area is not involved, direct vision is not diminished. No specific treatment has been devised.

Superficial Punctate Keratitis occasionally affects young persons who have

an upper respiratory infection, as tonsillitis. It may be a neuropathic manifestation, or it may be symptomatic of an endogenous infection, for inflammation of the uveal tissues often coexists. It must not be confused with "punctate keratitis." The disease begins with symptoms of conjunctivitis, and the circumcorneal vessels are engorged.

Small discrete spots are located in the epithelium, or in the superficial layers of the substantia propria beneath Bowman's membrane; they appear faint gray against the black pupil. By coalescence, groups or rows of lesions form. Fluorescein produces a light stain, as no vesicles are formed. The center of the cornea is generally affected, with a loss of luster, while the periphery may remain transparent. The lesion may be eccentric, but tends to encroach on the pupillary area. When this area is affected, vision is diminished. Faint lines may be traced in the cornea (see Fig. 105). A secretion may be present from the conjunctivitis.

The symptoms are diminished vision, lacrimation, and photophobia. The pain seems to be caused by a spasm of the sphincter of the iris which sharply contracts to light.

Treatment consists of atropin, hot applications for periods of ten minutes several times a day, and a pad to exclude light. As soon as the primary infection, as tonsillitis, begins to subside, yellow oxid of mercury in 1 or 2 per cent ointment is used. Salicylates are indicated throughout the course of the infection.

Keratitis e Lagophthalmo occurs in consequence of an inability to close the lids. The cornea is dry and clouded and becomes subject to infection. Ulceration or suppuration with extension to the deeper corneal tissues may follow. The condition may come on during long exhausting illnesses in which the orbicularis oculi relaxes, or in which corneal sensation is so obtunded that the winking reflex is abolished. In unconsciousness the cornea rotates upward; the lesion generally appears first in the lower part because it is most continually exposed. The condition is usually bilateral, except in facial paralysis.

Treatment is to keep the cornea clean and moist. White's ointment suffices for mild cases; the more severe ones require a pad or adhesive tapes to keep the lids closed, while the most severe require partial tarsorrhaphy. Ulcers and other complications need appropriate treatment.

Neuroparalytic Keratitis is due to paralysis of the trigeminus nerve from disease, and from the removal of or the injection of alcohol into the gasserian ganglion. It generally occurs in adults. Corneal sensation is abolished; the presence of a foreign body is not suspected and injury with infection may occur, lacrimation is diminished, and the impulse for reflex winking is bilateral from the healthy side only. The cornea is neither cleansed nor moistened.

Desiccation and desquamation of the epithelium begins in the pupillary area. If the epithelium is not regenerated the cornea flattens. Infecting organisms can colonize and propagate without resistance. The ciliary and conjunctival vessels are engorged. The ulceration progresses through the cornea and perforation occurs. The iris floats into the opening and is ultimately replaced by connective tissue. A corneal staphyloma (see Plate XII) or a keratocele may form. Very often the globe softens or shrinks and entropion develops. Vision is reduced. Symptoms are absent because of the anesthesia.

Treatment consists of protecting the cornea. When eye pads or adhesive tapes do not improve conditions, a median tarsorrhaphy ought to be done (an anesthetic is not necessary), and the lids should be left united for many months. Atropin and White's ointment are usually indicated. The progress is slow, and the prognosis is guarded.

Keratomalacia is a disease of the cornea due to impaired nutrition. It occurs in infants particularly, and in children below the age of five years. It accompanies malnutrition or marasmus following some of the acute exhausting diseases, or is due to syphilis, or is a consequence of the inability to get or to assimilate essential foods. Anemia is a common feature. While one cornea may be attacked earlier than the other, the disease is bilateral.

Xerophthalmia, a dry atrophy with shrinking of the conjunctiva, is a fairly constant complication; it has been observed in "epidemics" of keratomalacia due to deficiencies in the essential elements of a normal diet. The disease has been produced experimentally in animals by a diet deficient in fat-soluble vitamin A.

The cornea is insensitive, the child is apathetic, and the lids are seldom closed. These factors favor a rapid course. The part of the cornea exposed in the palpebral aperture is first attacked. There is a peculiar dry appearance, or a foam or froth may be present on the exposed part. Luster is diminished or lost. A haze of increasing density forms over the cornea, desiccation of epithelium occurs, infection is invited, ulceration and sloughing take place, and perforation with panophthalmitis may be expected. During the process the color becomes yellowish, and iritis with hypopyon is often consequent to the necrosis.

Night-blindness is an early symptom, but it may be unnoticed in very young patients. There is no real inflammatory reaction, and the usual symptoms are conspicuously absent. Constitutional symptoms are present, and alternating diarrhea and constipation are the rule.

Treatment is largely concerned with diet. Fresh whole milk is essential. Cod-liver oil is the most specific medicine. Chlorids have been recommended. Parsons approves the subcutaneous injection of large amounts of normal salt solution and the instillation of eserin. Dionin might be useful. Fairly hot

Plate IV.—Keratitis; Cataract.

A, *B* and *C*, interstitial keratitis in three stages, right eye. *D* and *E*, sclerosing keratitis, bilateral. *F*, cataract right eye; fundus left eye is shown in Plate IX. Distinguish opacities of the cornea and lens.

moist compresses are advised. Bandaging promises little, because the tears cannot reach the tissues through the unnatural secretions. The mortality rate is about 50 per cent.

INTERSTITIAL KERATITIS

Interstitial keratitis or parenchymatous keratitis is an inflammatory disease of the middle and deep layers of the cornea (Fig. 110). It is characterized by a round-celled infiltration, and the transparency of the cornea is diminished or lost.

The infiltration may begin in the center, or near the periphery and extend toward the center. The earliest sign is a faint localized gray veiling which obscures the details of the iris. The clouding involves most of the cornea, but it is not uniform in density. The cornea has a whitish appearance and the luster is lost.

Fig. 110.

New vessels from the anterior ciliary arteries invade the substantia propria and branch freely but do not anastomose. They are usually arranged in bundles and have a brush- or broom-like appearance (see Fig. 106). Being deeply covered the pannus appears pink and is called a "salmon patch." These vessels may extend as far as the center of the cornea.

Vessels from the marginal loops invade the superficial tissues. They are red, straight in their course, and extend on to the surface only a short distance. They disappear after a brief existence. As healing takes place the deeper vessels lose their color; but as they are replaced by connective tissue the cornea does not fully regain its transparency (see Fig. 105).

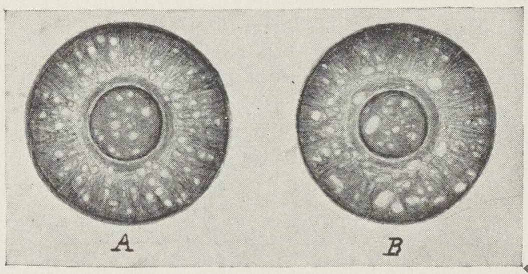


Fig. 111.—Keratic Precipitates.

The cornea tends to clear from the periphery toward the center (Plate IV, Fig. C). When the pupillary area fails to become clear vision is permanently damaged. The opacity may be so dense that it resembles porcelain, or only a faint haze may remain.

When the uveal tissues are involved, leukocytes and exudates migrate into the anterior chamber. These products form in clumps which adhere to the posterior surface of the cornea; they are called "mutton fat" deposits, keratic precipitates and k.p. (Fig. 111). With focal illumination and magnification they are visible, except when a dense corneal infiltration conceals them. The

anterior chamber is usually deep. When the ciliary body is inflamed the secretion of aqueous is diminished and the tension of the eyeball is lowered.

The tonometer ought to be used sparingly in cases of corneal disease. Taking the tension with the fingers will suffice. Because of exceptional cases in which there is a rise of tension, the physician should be on the alert.

The symptoms are photophobia, increased lacrimation, pain and blepharospasm, and they vary according to the severity of the inflammation and the age of the patient. Both eyes are nearly always affected, especially in younger children, but one in advance of the other. Iritis and iridocyclitis are common complications. The acute symptoms last from six to ten weeks, but the disease does not disappear for a year or more. During the period of recovery the cornea may become thin, and keratectasia may be expected. Rarely, buphthalmos has followed the disease.

Interstitial keratitis generally occurs in children between the ages of five and fifteen years, although it has been recognized in infants and in patients in the fourth decade of life. It is more frequent in girls than in boys. Congenital or hereditary syphilis is responsible for 90 per cent of the cases, and tuberculosis for most of the remainder. Inherited syphilis and tuberculosis can coexist. While the Wassermann and luetin tests are rather uniformly positive in these patients, there are certain clinical features that are even more important.

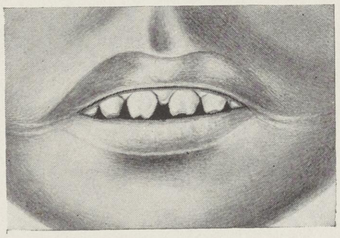


Fig. 112.

Any two or more of the following peculiarities are presumptive in the diagnosis of inherited syphilis: Prominent frontal eminences, saddle-nose, flat face, scars at the angles of the mouth from rhagades, shotlike painless posterior cervical glands, disease of the lacrimal sac or nasolacrimal duct especially when the secretions from them have the odor of necrosing bone, tibial tophi, chronic synovitis of both knees and vertically oval pupils with a history of syphilis in the parents.

Of greater significance are Hutchinson's teeth (Fig. 112). The upper central incisors of the second dentition are usually deformed by vertical concavities in their biting edges. This defect is generally apparent to inspection. If not apparent, the teeth should be transilluminated; marked thinning that corresponds to the concavities described is suspicious. In other patients the teeth will be peglike, poorly developed in all dimensions and widely spaced. Otosclerosis or manifest deafness is not uncommon. Many of the children are either precocious or dull, but not normal in mental development. The classical diagnostic triad consists of interstitial keratitis, Hutchinson's teeth and otosclerosis.

It is claimed that interstitial keratitis can be developed from acquired syphilis; but it does not seem to have been proven that hereditary syphilis did not already exist in these patients. In this type the disease is usually limited to one cornea, and it has been observed in many instances that an injury had been sustained by the affected eye. Spirochetes have been found in the cornea both of fetal and enucleated infant eyes.

In general, the older the patient the better the prognosis. Ulceration, abscess or perforation of the cornea does not occur in uncomplicated cases. As the disease is more prone to appear in sickly or undernourished children, such individuals are least able to combat the attack and regain useful vision. When these patients are also subjected to neglect in care or treatment, the prognosis is correspondingly more grave.

Treatment is both local and general. Nutritious diet, fresh air and sunshine, regular habits, general medication, and antisyphilitic treatment are all of value. Children do well on biniodid or bichlorid of mercury, or mercury by inunction, and potassium iodid. Approved arsenicals are used intravenously. Treatment does not prevent the fellow eye from becoming involved.

Local treatment avoids irritating agents while the disease is active. Dionin is indicated to relieve pain and encourage lymph movement. Moist heat in the form of compresses is agreeable and helpful. Dark glasses must be worn when in bright light. Atropin is indicated except when the tension is increased. When atropin solutions fail to dilate the pupil it is advisable to use dry atropin, preceded by 2 or 3 drops of a 4 per cent cocain solution. It may be given subconjunctivally also.

When the disease begins to subside, yellow oxid of mercury 1 or 2 per cent ointment is used with massage; atropin may be combined with it. This should be done daily if well tolerated. Subconjunctival injections of normal salt solution are often helpful. These measures are designed to clear the opacity. Dionin may be continued, with intervals of rest.

Keratectasia, when recent, requires a pressure bandage. For persistent central opacity an optical iridectomy may be performed, but not until all probable clearing can be dismissed. This is made in the lower nasal quadrant of the iris, unless another part of the cornea is much clearer.

Interstitial keratitis may be due to tuberculosis when no evidence of syphilis is found. It may be secondary to tuberculosis of the iris or ciliary body, or associated with tuberculous infections of the conjunctiva, sclera or pectinate ligament. Small yellowish nodules appear near the margin of the cornea, and corresponding to their locations, the ciliary vessels are engorged. These nodules must not be confused with gummata of the sclera. The treatment consists of tuberculin, and the local measures already specified.

In interstitial keratitis the cornea is sometimes marked by parallel and branching lines that arise at the margin of the cornea and taper toward its center (see Plate IV, Fig. A). They may be of single or double contour, and are usually horizontal. Somewhat similar lines appear in *superficial linear keratitis,* but they are vertical, taper at the ends, do not branch, and have nodes which stain with fluorescein. Another form is called *lattice-shaped* or *letter-shaped keratitis,* in which the lines are crossed. Both forms are characterized by pain and congestion and both are rare.

KERATITIS, DEEP FORMS

Keratitis Profunda is a slowly developing centrally located interstitial keratitis which attacks adults; it is usually unilateral. The middle and deep layers of the cornea are affected. It may follow injury to the cornea, or accompany colds, focal infections, malaria, herpes zoster ophthalmicus, tuberculosis or uveitis. The symptoms are usually mild, but may be moderately severe. Circumcorneal congestion and pannus seldom occur. The lesion heals without ulceration, and with or without a faint opacity. The local treatment is like that for interstitial keratitis. The duration is from a month to a year.

Keratitis Marginalis Profunda attacks one eye in elderly people, without antecedent scleritis. A grayish-yellow infiltrate occupies the zone between the sclera and the arcus senilis. A part or all of the circumference may be attacked. Healing occurs in a week or two, but a permanent gray opacity remains. The treatment is symptomatic.

Marginal Atrophy of the Cornea occupies the same location. The tissues become attenuated and a peripheral ectasia may occur. This causes corneal deformity and astigmatism. No treatment is indicated, except lenses to correct the astigmatism.

Epithelial Dystrophy of the Cornea occurs in the aged, and one or both eyes may be affected. It somewhat resembles the hazy cornea of glaucoma. The opacity is most pronounced over the pupillary area, and vision is diminished. Vesicles may form. The cornea is insensitive, and symptoms are absent. No effective treatment is known.

Zonular Keratitis occurs in both eyes of elderly people. It occupies an area corresponding to the exposed space between the lids. While it is more commonly found in blind eyes and shrunken globes, it may affect functionally good eyes. The opacity is gray or yellowish brown and rough. It is the result of hyalin degeneration and the deposition of calcium salts, first in Bowman's membrane, and later both beneath and upon it, so that the epithelium is affected. A narrow band of clear cornea separates it from the limbus.

The change begins in both the nasal and temporal areas, and gradually extends toward the center; the pupillary area may remain free for a long time. It is believed to be due to nutritional deficiencies. The gritty material can be removed by gentle scraping or curetting. In appearance the lesion resembles *lead incrustations* that form from the use of lead lotions in conjunctivitis; lead incrustations are usually white.

Sclerosing Keratitis affects the substantia propria, which is anatomically continuous with the sclera; it does not refer to a process of sclerosis. The opacity is continuous from the sclera, and invades the cornea in a triangular shape with a rounded apex directed toward the pupil. It is usually multiple, but the pupillary area generally escapes. It may arise from a scleritic nodule. The causes are the same as those which produce scleritis: Syphilis and tuberculosis in young, and focal infections in older patients. Iritis and cyclitis are sometimes associated. Vascularization and ulceration are rare, but the opacity seldom disappears.

The opacity begins as a gray or yellowish gray, becomes white, and when healed has the bluish white color of natural sclera. It may eventually look like porcelain (see Plate IV, Figs. D and E). The course is chronic and is characterized by relapses; symptoms of irritation may be marked or absent. The treatment is directed to the underlying disease. When irritation is severe it should be treated like interstitial keratitis. Mild cases need no local treatment.

Arcus Senilis or *gerontoxon* consists of a white band about 1 millimeter broad which is separated from the sclera by a band of clear cornea of equal breadth. It extends deeply into the substance of the cornea. The outer border is sharply defined, while the inner fades into clear cornea. The eyes are equally and symmetrically affected as a rule. It rarely appears before late middle life, and is rather constant in the aged.

The opacity is established without previous disease, and has no recognized clinical significance. It is usually ascribed to deposits of minute fat globules in, or to fatty degeneration of, the substantia propria outside the limits of Bowman's membrane. It forms first above, then below, and eventually the circle is completed; the older portions are broader than the newer.

Arcus Juvenilis or *embryotoxon* is a rare congenital shallow arcuate opacity resembling arcus senilis. It may be found singly, or on opposite sides of the cornea, as the "upper nasal and the lower temporal," or the "medial and lateral," but never "above and below"; a complete circle is seldom seen. Both margins of the opacity are sharply defined. Both eyes are seldom affected.

ULCERATION

Ulcus Serpens, serpiginous or pneumococcal ulcer (Plate V) develops in a wound of the cornea. The nearer a wound is to the central area of the cornea, the more prone it is to become infected, and the more devastating are the consequences. The proximity of the corneal arterial loops enhances the effectiveness of defense processes when the infection occurs near the periphery. The ulcer creeps toward the center of the cornea more than it does toward the margin, until the toxins are produced in sufficient quantities to overcome leukocytes; then the ulcer spreads toward the periphery also. The lesion tends to encircle the center, even while the advancing zone widens.

While the encapsulated diplococcus of Fränkel-Weichselbaum is the usual organism present, others may be found also, either coexistent or independent. A large number of these patients have a chronic dacryocystitis. The pneumococcus is practically always present in the latter disease. When a corneal injury is found it is good practice to examine the tear sac; if it is diseased it can be extirpated or the puncta can be sealed with the galvanocautery at once.

Any purulent conjunctivitis can supply the organisms to complicate a corneal injury. Diphtheritic and gonorrheal inflammations of the conjunctiva seem to have the ability to infect the cornea without a previous injury, but they seldom produce a serpent ulcer.

The conjunctival sac communicates with the nasal fossa by way of the nasolacrimal duct. Attention to nasal conditions is advisable in the treatment of ulcers of the cornea. Atypical serpent ulcers may occur during the course of any of the exanthemata, especially measles; in these instances the ulcer ordinarily appears late in the course of the antecedent disease.

The typical pneumococcal ulcer occurs in adults, particularly among the working classes. The latter neglect their supposed minor injuries, and are disposed to seek medical aid only when they are incapacitated. They are subject to ulcer because of their exposure to injury, their disposition to diseases and deformities of the nasal structures with consequent inflammations of the tear passages, and their liability to conjunctival disease. They wipe their eyes with soiled handkerchiefs, hands, gloves, sleeves or what not, and thus contribute to the opportunities for infection.

A recent injury of the cornea will stain with fluorescein. If healing is prompt, no staining will be seen after twenty-four or forty-eight hours. When the stain is accepted after a period of forty-eight hours and has a fresh green appearance, it is probable that infection has occurred. Inspection reveals a faint grayish nebula at the bottom of the wound. When this is examined by

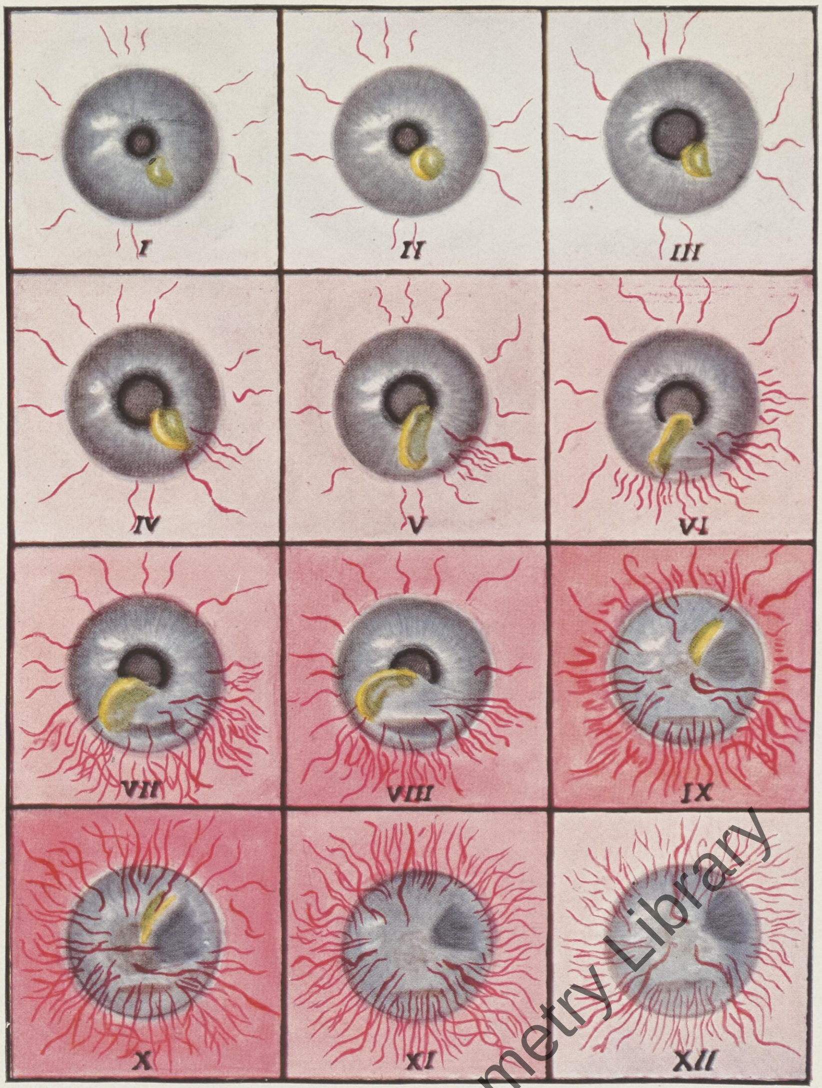


PLATE V.—ULCUS SERPENS, INFECTION OF LACRIMAL SAC. INJURY TO CORNEA ON APRIL 11, 1923.

I, staining, third day; II, invasion, eighth day; III, extension, ninth day; IV, pannus forming, tenth day; V, healing in wake of ulcer, eleventh day; VI, hypopyon, fourteenth day; VII, engorgement of conjunctival vessels, fifteenth day; VIII, paracentesis, sixteenth day; IX, conjunctival chemosis, twenty-first day; X, lens delivered spontaneously through rupture of cornea, twenty-fourth day; XI, cornea healing, twenty-eighth day; XII, cornea healed, sixty-seventh day. (See Plate VI, not the same patient.)

focal illumination and magnification, it will be observed that fine lines radiate from the wound into the substantia propria beneath Bowman's membrane. Outside this area the cornea is normal in appearance.

The lines of the opacity suggest a local arrest of the circulation of fluids. This is caused by the action of the toxins of the invading organisms on the cells of the cornea. A region of corneal stasis becomes necrotic quickly. The toxins prepare the tissues for the attack of the organisms. In serpent ulcer the invaders advance along one front. As the leukocytes enter this zone of preparation, they are overcome, die in masses and become opaque. This gives the appearance of an infiltration in front of the necrosing process; it is visible to inspection.

The circumcorneal vessels and those of the ocular conjunctiva are engorged. The whole anterior surface of the eye now has the appearance of a very active inflammation. Branches of vessels cross the border of the cornea and enter the ulcerating area behind the advancing organisms. The ferment action of the white blood-cells digests the necrotic mass. The floor of the ulcer is cleansed and prepared for the proliferation of connective tissue. The process of repair follows in the path of the process of destruction. This amounts to preservation of the globe only.

Vision cannot be regained, once the pupillary area of the cornea has been invaded by pneumococcal ulcer, for two reasons. (1) The thickness of the cornea is not restored, and the surface is permanently and irregularly flattened. (2) The repair consists of opaque connective tissue. The cornea loses its transparency and its smooth surface; it is optically defective.

As the ulcer advances and repair follows, new blood-vessels cross the limbus to engage in mending the damaged tissues. These vessels are mostly permanent, but they undergo a partial sclerosis after healing has been completed. This is more noticeable in the larger vessels, for the smaller ones are usually obscured by connective tissue.

The mechanism for the formation of pus and hypopyon in the anterior chamber has been noticed. In serpent ulcer the same mechanism explains other phenomena. All the systems of vessels supplying the cornea participate in the vascular invasion. As the deepest of these are derived from the vessels which also supply the iris and ciliary body, they are likewise engorged because of the attraction of leukocytes to an infected cornea.

Masses of exudate derived from the uveal vessels are attracted toward and cling to the posterior surface of the cornea, especially behind the most active part of the ulcer. Sometimes this can be seen by looking from the side through clear cornea at its posterior surface. The exudate is composed of pus cells and fibrin; ferment is liberated and the endothelium is destroyed (Fig. 113).

When Descemet's membrane yields, the pus comes into contact with the posterior layers of the substantia propria and an ulcer is formed on this surface also.

Being attacked from both surfaces, the cornea succumbs at this point and perforation occurs. Either the iris or the lens is brought against the opening by the escape of aqueous humor. Where the pupil has been properly dilated, the lens is almost sure to be lost through a central opening. Perforation is attended by great relief from pain, and oftentimes by a temporary or permanent cessation of all inflammatory symptoms. Shortly before perforation occurs, pain is especially intense.

Where the lens is lost and the iris does not float into the wound to close it, intra-ocular infection may be expected. If such infection does not occur, the closing of the aperture will depend upon the proliferation of connective tissue.

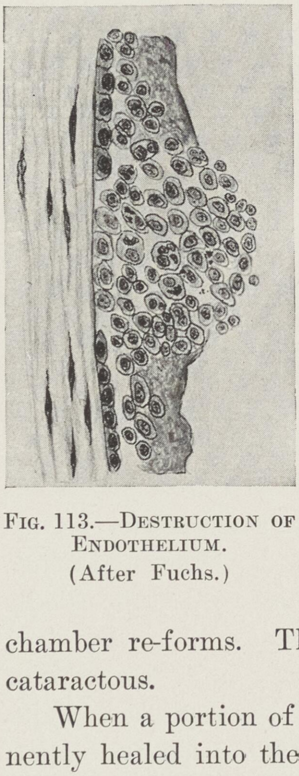


Fig. 113.—Destruction of Endothelium. (After Fuchs.)

When the intra-ocular pressure rises before the reparative tissue is strong, a secondary rupture may occur. The constant trickling of aqueous through this wound keeps it open. Where epithelium grows into the channel a fistula is formed. A pressure dressing should be applied early. Later a conjunctival flap can be used to close it. When increased tension is present for some time, perforation may be accompanied by intra-ocular hemorrhage or detachment of the retina. In such instances the eyeball may gradually soften and shrink.

Where the opening is small the lens will block it. Healing takes place with the capsule of the lens in contact with the new tissue (see Plate VI). The anterior chamber re-forms. The lens is torn from its corneal adhesion and becomes cataractous.

When a portion of the iris occludes a perforation wound, it will be permanently healed into the cicatrix. Connective-tissue cells gradually replace the cells of the iris that are in contact with the healing cornea. The point of adhesion will be marked by a dense white spot, an adherent leukoma. This may be tattooed if desired.

Although pneumococci are not found in the hypopyon or in the aqueous previous to perforation, a true iritis or iridocyclitis often complicates cases of serpent ulcer. Sometimes the iris adheres to the capsule of the lens at one or more points, or without interruption around the entire circumference of the pupil, and forms posterior synechiæ or annular synechia respectively. In

annular synechia the aqueous cannot enter the anterior chamber, and the iris is bowed forward, iris bombé (see Fig. 138).

In other instances the iris adheres to the posterior surface of the cornea at some point and forms an anterior synechia, with or without perforation. After perforation, the iris may flatten against the cornea and remain there with or without the formation of a staphyloma; the globe will soften. Occasionally staphyloma and scondary glaucoma precede softening (see Plate XII). In the absence of increased tension the cornea flattens.

Not infrequently the physician will first see the patient after the ulcer is established. Diagnosis will depend upon a knowledge of the course of the ulcer and its developmental stages.

A disk-shaped opacity is found in the cornea, usually nearer the center than the periphery. The central area of the disk is comparatively pale, while one margin is denser and of a white or creamy color which fades out as an infiltration advancing before the ulcer. The dense margin is crescentic or horseshoe shaped. A deeper opacity at one segment of the crescent indicates the probable direction of the most intense activity. Where necrosis has already begun, it will be seen as a yellow crescent; with fluorescein the ulcer stains green, and the necrotic tissue yellowish.

The floor of the ulcer is depressed below the level of surrounding uninvaded cornea. The depression stains less intensely because in that area the disease has spent its force and repair has begun. The cornea loses its luster, the iris is swollen and discolored (iritis), the aqueous is cloudy and hypopyon is present as well as general conjunctival congestion.

The ulcer enlarges in the direction indicated by the denser opacity, and extends more deeply into the corneal tissues. Pus cells coat the floor of the ulcer. The epithelium is not attacked directly, for the organisms advance and propagate beneath a ledge of epithelium. Smears made from the floor of the ulcer seldom show pneumococci; scrapings should be made from beneath the shelf. It is necessary to consider this undermining in treating the ulcer, otherwise the organisms are not reached.

This disease is sometimes called *hypopyon ulcer,* but the designation is too broad. The position of an ordinary hypopyon (see Fig. 107) is changed by gravity. The hypopyon of serpent ulcer is held in one position by the fibrin content. The exudate is drawn to the cornea in clumps of various sizes; the smaller and lighter adhere higher, the larger and heavier lower on the cornea, so that the upper border is convex or pyramidal (Figs. 114 and 115). The height of the pyramid often indicates the severity of the reaction.

The exudate assists in the formation of anterior synechiæ by drawing the iris toward and attaching it to the cornea. An adherent leukoma might form

from this process, but it is more likely to occur where the iris is incarcerated in a perforation.

Symptoms are pain, lacrimation, photophobia and blepharospasm. To these are often added headache, insomnia, loss of appetite and debility.

Prognosis is bad for vision, and guarded for preservation of the eyeball. A few cases recover and retain some useful vision. The prognosis is better when some organism other than the pneumococcus is responsible; the others do not produce typical serpent ulcers.

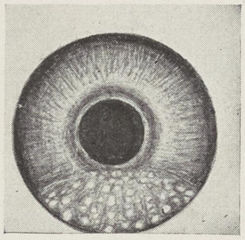


Fig. 114.

Atypical forms must be studied bacteriologically to identify the active organism. The diplobacillus of Morax-Axenfeld and that of Petit require treatment by zinc salts in addition to other measures. The *Bacillus pyocyaneus* usually produces a malignant process; the lesion is deep seated, lacks the crescentic advancing zone, and contains pus (de Schweinitz). The pyogenic cocci and mixed infections also produce atypical ulcers.

Treatment must be instituted promptly and energetically since the ulcer quickly becomes intractable. Because of the frequency of tear sac infections the puncta should be sealed with the galvanocautery, and the sac may be extirpated if diseased. The conjunctival sac, including the fornices, should be irrigated daily with an antiseptic solution, such as 1: 6,000 to 1: 8,000 bichlorid of mercury. Phenacain in 1 or 2 per cent solution or ointment is indicated for pain; dionin 1 to 5 per cent may be added to it when needed. White's ointment is applied to the conjunctival sac at each dressing.

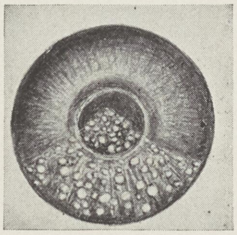


Fig. 115.

Atropin sulphate one drop of 1 per cent solution is instilled at hourly intervals until the pupil dilates, and then with sufficient frequency to maintain dilatation. This reduces ciliary congestion and soothes the irritation in the uveal tissue. Although there may be a tendency toward glaucoma, existing iritis is the more important consideration. Atropin is seldom contra-indicated. When a prospective perforation is nearer the limbus than the center of the cornea, it is within the discretion of the surgeon to omit atropin, and to use pilocarpin 1 per cent or eserin 0.25 per cent to contract the pupil and draw the iris back from contact with the cornea.

Between treatments the eye must be protected with a dressing. Where there is much conjunctival secretion, especially if purulent, the dressings consist of layers of gauze kept saturated with 1: 10,000 mercuric chlorid solution. Such patients do better when hospitalized and attended by an ophthalmic nurse.

Heat is applied by moist compresses for fifteen minutes of each hour or two hours. If heat is disagreeable, iced compresses may be tried, but only for five minutes of each hour.

Optochin, or ethylhydrocuprein hydrochlorid is said to be specific for pneumococcal ulcer. It is applied to the ulcer in 1 per cent solution, and instilled in 0.5 per cent solution every two hours. Mercurochrome 220 in 1 or 2 per cent solution is useful. It stains the ulcer, supplants fluorescein in this respect, and is a superior antiseptic. Mercurophen 1:15,000, and metaphen 1:5,000 have both been recommended for irrigations. The silver salts are useful in cases of purulent conjunctival secretion. Zinc sulphate in 0.5 to 1 per cent solution has a specific action on the diplobacillus of Morax-Axenfeld and on that of Petit. It is applied frequently in addition to routine treatment.

The ulcer is attacked at its advancing edge, and beneath the overhanging ledge (Fig. 116). Some surgeons excise the shelf and others curette or cauterize beneath it. Cauterization is done with liquid phenol, tincture of iodin, the actual or galvanocautery and strong solutions of zinc sulphate. Good light is necessary. The cornea is anesthetized, the lids are separated with the speculum, the globe is held with fixation forceps and the cautery is applied.

Fig. 116.

The tip of a sharpened toothpick, match or wooden splinter is dipped into pure phenol and allowed to absorb what it can. When sure that no excess of acid clings to the wood, the point is inserted beneath the ledge and swept along the length of the advancing crescent and across the floor of the ulcer. The tissue turns white. The treatment is repeated every one to three days as indicated.

Tincture of iodin is used; a bit of cotton is wound tightly on the end of a sharpened toothpick; this end is dipped into the tincture, and the alcohol is allowed to evaporate. The treatment is executed as with phenol. The reaction is severe. Phenol is preferable because it is anesthetic and penetrates more deeply and widely into the tissues. Sterile 20 per cent solution of sulphate of zinc is used in the same manner as tincture of iodin.

The actual cautery is less satisfactory than the galvanocautery; either can be used by bringing the tip to a dull red heat, and using it in the same way that the chemicals would be used. The galvanocautery can also be used by inserting the cold tip beneath the shelf, closing the current, and withdrawing the instrument while it is still hot. By repeated applications at successive points the entire crescent can be treated. If the tip is allowed to cool while in contact with the tissues it will stick to them.

Good effects sometimes follow radiation of heat from the tip while it is held near to but not in contact with the ulcer. The thermophore enables the

operator to use a known degree of heat. The pasteurizer was designed for heat radiation, but the galvanocautery tip serves the purpose. Currents of hot air have been used.

Antipneumococcic serum has not given uniform results either when given hypodermically or when applied direct to the ulcer. The parenteral injection of boiled milk and the administration of antityphoid vaccine and antitetanic serum as nonspecific proteins have given results that encourage further study. Key emphasizes the advantages of antidiphtheritic serum, because it can be used in exact doses; he recommends that it be given at once after cauterizing the ulcer.

Paracentesis is made usually in the lowest part of the cornea. The aqueous is allowed to escape slowly. The hypopyon may be teased out with a fine needle. The wound is opened each day by depressing its lower lip. This procedure usually has a good effect.

A Saemisch section is performed with a cataract knife. The puncture is made in clear cornea in *front* of the midpoint of the advancing crescent, the counterpuncture is made in clear cornea *beyond* the ulcer, and the knife is made to cut its way out. Injury to the iris and lens must be avoided. Intense pain occurs when the aqueous gushes out. For that reason a general anesthetic is often preferred to a local. The wound should be opened daily. The good effects of this operation are immediate and almost certain.

The general physical condition of the patient must be maintained at a high level.

MISCELLANEOUS DISEASES AND DEFORMITIES

Ulcus serpens in early stages may need to be differentiated from disk-shaped keratitis, keratitis profunda and keratomycosis.

Disk-shaped Keratitis is thought to be due to infection following some injury such as a blow; it develops rapidly like serpent ulcer. The opacity is situated in the middle layers of the central area of the cornea. A dense center indicates the point of infection, and a peripheral zone of lighter density outlines its extent. The border is evenly curved, and has no yellowish crescentic advancing edge. Little destruction of tissue, but permanent opacity is to be expected.

The course extends over several weeks. Atropin, dionin, subconjunctival injections of normal salt solution, White's ointment and bandage are indicated. Massage with 1 or 2 per cent yellow oxid of mercury ointment is of service after the acute symptoms have subsided; this must not be used at the *same time* as dionin.

Keratomycosis is caused by a fungus that has been introduced into injured

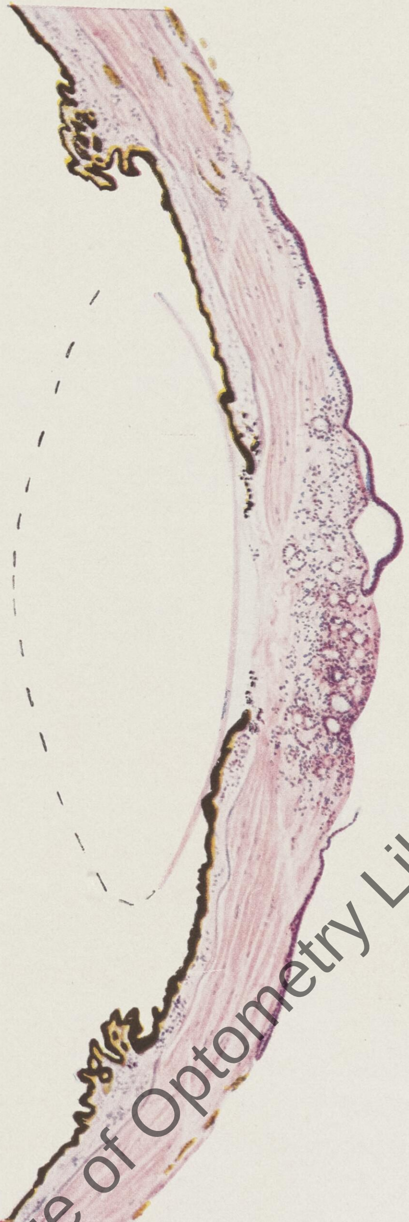


PLATE VI.—SECTION OF CORNEA PERFORATED BY ULCER.
The lens crowded the iris against the cornea; healing process.

corneal epithelium. An infiltration occurs in the central area of the cornea; this is followed by disintegration, necrosis and sequestrum formation. The lesion is bounded by a line of demarcation and has a dry uneven surface. Hypopyon is often present, but irritation is not marked and there is no advancing edge.

The sequestrum separates, and a permanent loss of tissue with opacity marks the site. A cicatrix forms on the floor of the cavity. In especially severe cases the cornea may be perforated. The fungus is identified in scrapings under the microscope. The treatment is to remove the plug of necrotic tissue and cauterize the base and edges of the ulcer with phenol or the galvanocautery.

Mooren's Ulcer, chronic serpiginous ulcer or rodent ulcer of the cornea, attacks the superficial layers near the margin and progresses toward the center. Blood-vessels follow in the path of the ulcer as in phlyctenular keratitis, but the ulcer is less painful than the keratitis. It is not purulent, and is not to be confused with serpent ulcer.

The lesions occasionally coalesce as they approach the center of the cornea. As the process undermines the epithelium, there is an overhanging shelf marked by a faint line at the advancing edge. The lesions appear to heal, but suddenly revive and renew the advance. This is a characteristic feature of the disease. The entire corneal surface will have been affected eventually with serious impairment of vision.

Treatment.—Phenol and the galvanocautery may be used as in serpent ulcer. Absolute alcohol may be used in the same way as phenol. Dionin in 1 to 5 per cent ointment has seemed to be of value. Elderly people are subject to the disease, and their general health demands attention.

Secondary Ulcers of the Cornea occur in many varities of keratitis, such as dendritic and phlyctenular, in purulent conjunctivitis, such as gonorrheal, in trachoma, iritis, endogenous infections, and in impaired health. The primary disease must be treated. Atropin, dionin, antiseptics, cautery, canthotomy in trachoma, and so forth are employed as indicated. Many rare named forms of ulcer are described in the literature, but their inclusion here is not practicable.

Opacities generally remain after ulceration of the cornea; they are disfiguring and their removal is difficult or impossible. When situated over the pupillary area, vision is impaired. The surface of the cornea is often so uneven that no lens can correct the irregular corneal astigmatism. In such cases an optical iridectomy is without effect. It is of service where the contour of the cornea is regular. Disfiguring central opacities can be tattooed to resemble a pupil. Recent opacities sometimes fade under dionin and subconjunctival

injections of normal salt solution continued over long periods of time. These measures are indicated where the fellow eye is blind or nearly so.

Birth Injuries of the cornea result from prolonged labor, faulty application of forceps, or rough handling. These are usually manifested by opacities, which are diffuse when due to edema from contusions and linear where Descemet's membrane has been ruptured. Other opacities, classed as congenital, are due to intra-uterine ocular inflammations like interstitial keratitis or gonorrheal ophthalmia. In interstitial keratitis in the newborn the cornea may be vertically oval or egg-shaped.

Melanosis is a congenital pigmentation of the cornea.

Sclerosis is a peripheral annular opacity that is said to be due to a failure of the scleral layer of the cornea to become transparent.

Cornea Plana signifies that the cornea has the same radius of curvature as the sclera.

Megalocornea denotes a large cornea.

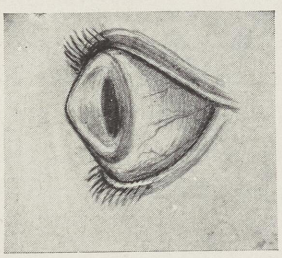


Fig. 117.—Keratoconus.

Microcornea denotes a small cornea.

Tumors of the Cornea occur at the limbus and are derived from conjunctival, scleral or uveal tissue. They are so rare and of such wide variety that no attempt is made to discuss them here. They are to be excised, and the site exposed to radium under expert supervision.

Keratocele or descemetocele is formed by the destruction at some point of all of the layers of the cornea except Descemet's membrane and the endothelium; these protrude into or through the crater. Touching the protrusion with a probe will reveal its character.

Keratoconus or conical cornea is a noninflammatory protrusion of the cornea in the form of a cone. It is usually bilateral. It may be recognized when looked at from the side (Fig. 117), or with Placido's disk. It occurs most frequently between the ages of twelve and twenty years, and is due to a progressive thinning of the corneal tissues. Periods of alternating opacity and transparency are to be expected. Myopia or myopic astigmatism is present. Congenital keratoconus or anterior staphyloma have been observed.

The treatment of keratoconus consists of keeping refractive errors fully corrected, the avoidance of habitual or steady use of the eyes for near work which demands accommodative effort, and attention to the general health. Pilocarpin may be used routinely to facilitate aqueous drainage and maintain a low intra-ocular tension. Sometimes the deformity requires surgical correction.

Keratectasia is a protrusion which follows a keratitis that destroys some of the corneal layers (Fig. 118). It is opaque because connective tissue has pro-

liferated in the cornea during the healing process; otherwise it resembles a keratoconus. No iris tissue is incorporated in it.

Keratoglobus designates the noninflammatory uniform enlargement, such as seen in buphthalmos.

Staphyloma of the Cornea often follows a perforation whether from injury or ulceration. The iris lies against the wound and is bound to the posterior surface of the cornea by inflammatory adhesions (see Plate XII). In healing, connective tissue replaces iris tissue with consequent opacity. Some staphylomata protrude between the lids and prevent their closure. They become painful. A conjunctival flap is formed, either for a purse-string suture or as an apron, and silk sutures are inserted. The staphyloma is removed and the conjunctival flap is applied. (See page 67.)

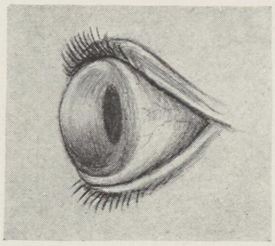


Fig. 118.—Keratoglobus.

The alternative operation is enucleation, for the cosmetic result of the flap method is not desirable in all cases. The stump formed by excising the cornea will support an artificial eye, and shrinking of the orbital tissues may not occur as it often does following enucleation. These eyes are generally deformed in consequence of intra-ocular inflammation; they react to irritation and may not support a prosthesis. An irritable eyeball should be removed.

When a keratocele ruptures it is converted into a staphyloma by the obliteration of the anterior chamber. An imminent rupture may be averted by a pressure bandage or a conjunctival flap that covers the defect in the cornea. Atropin is indicated to draw the iris away from the site of a threatened central rupture, and a miotic is indicated where the defect is peripheral.

CHAPTER VI

THE SCLERA

ANATOMY AND DISEASES

The posterior five-sixths of the outer coat of the eyeball is the sclera, sclerotic coat, or "the white of the eye." It is flexible but not elastic. It is continuous with the substantia propria of the cornea and is similarly constructed of flattened bundles of fibrous tissue which are arranged in laminæ; the bundles form angles with each other. The sclera is about 1 millimeter thick posteriorly, 0.5 millimeter at the equator of the globe, and 0.6 millimeter between the insertions of the recti muscles and the cornea. The tendons of the extra-ocular muscles are inserted into the scleral fabric.

There are several openings for the passage of nerves and blood-vessels. The optic nerve passes through the scleral foramen. The ciliary arteries and nerves and the venæ vorticosæ pass through oblique channels called emissaria. The anterior ciliary vessels pass through perforations in front of the insertions of the recti muscles (see Fig. 82). The sinus venosus scleræ or Schlemm's canal lies in the wall of the sclera immediately behind its junction with the cornea.

The sclerotic coat is composed of three anatomical layers. The episclera, a part of the fascia bulbi, is outermost; the principal part is the sclera proper, and the lamina fusca is innermost, next to the choroid. The episclera is plentifully and the sclera is scantily supplied with blood-vessels. Sensation is by way of the ciliary nerves to the trigeminus. The section anterior to the equator is the more susceptible to disease, which usually is of an inflammatory character. If superficial it is episcleritis; if deep it is scleritis. Each form is subdivided into varieties.

Episcleritis is marked by a circumscribed purplish patch, flat or raised above the surrounding level, with engorgement of the vessels traversing it. It may or may not be tender and painful. As it does not invade quite to the cornea (Fig. 119), the conjunctiva can be moved over it freely. The color is largely due to the engorgement of the deeper vessels, in which the blood columns are obscured by overlying tissues.

The disease is characterized by recurrences in new locations until the cornea has been encircled. The duration of each attack is from two to eight weeks, and the site is generally marked by a permanent dark discoloration. Focal

infections are often present, although syphilis and tuberculosis are not infrequently associated. Both eyes may be attacked.

Treatment is symptomatic and empirical. Dionin and subconjunctival injections of normal salt solution are used. Moist hot compresses are useful for pain. Atropin is indicated for ciliary congestion. Yellow oxid of mercury 1 per cent ointment may be used with massage through the closed lid if tenderness will permit. Large doses of sodium salicylate or aspirin, and potassium iodid are usually beneficial. Free saline catharsis is nearly always indicated. Syphilis and tuberculosis require treatment. Suppuration does not occur.

Episcleritis Periodica Fugax is characterized by engorgement of the vessels with diffuse edema. Successive portions of the anterior sclera are subject to attack. The eye is distinctly red, and the attacks recur with a tendency toward regularity. It resists treatment obstinately. Quinin and the measures outlined for scleritis may be tried.

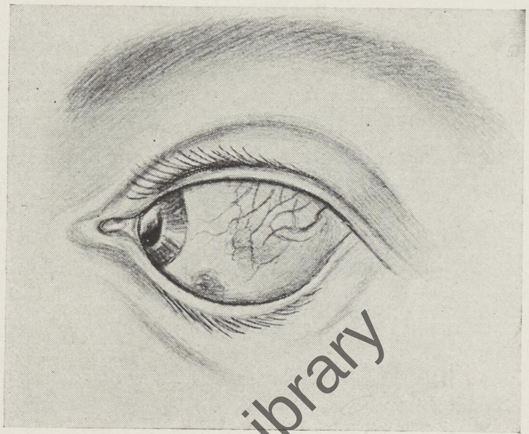


Fig. 119.—Episcleritis. (After Fuchs.)

Scleritis is an inflammation of the sclera proper; it has the same etiology as episcleritis. It differs from the latter in its course and effects. It tends to affect other ocular tissues. During the subsidence of an attack the affected sclera is attenuated; in time this is demonstrable as an ectasia or a staphyloma. Successive attacks may produce these until the cornea is surrounded by them. The ectasiæ mostly form in the ciliary zone, and have a bluish appearance because the uveal pigment is not concealed by the thin wall of the stretched sclera. Both eyes may be affected.

Complications consist of sclerosing keratitis (see Plate IV, Figs. D and E), corneal infiltration, iritis, choroiditis, vitreous opacities, and axial myopia. The complicating inflammation in intra-ocular tissues causes a secondary glaucoma, and the raised tension produces the ectasiæ and staphylomata. The treatment for uncomplicated scleritis is like that for episcleritis. The disease is progressive and exceedingly obstinate. Complications require treatment.

Brawny Scleritis, gelatinous or annular scleritis, attacks older persons. A brownish colored conjunctiva with very prominent widely dilated blood-vessels covers the globe from the cornea to the fornices. The edema does not suggest thin fluids, but rather a fixed semifluid mass of the consistency of gelatin. Intra-ocular complications usually destroy vision, and both eyes are liable to

attack. No local treatment has modified the disease to any extent; the general health should be given attention.

Syphilis of the Sclera occurs in the form of gumma. A yellowish swelling arises near the cornea, and intra-ocular complications may be expected, particularly where the gumma extends backward toward the equator.

Tubercle of the Sclera occurs in the same situation, but can usually be traced directly from the iris or ciliary body.

Tumors of the Sclera are rare, but the tissue is subject to fibroma and sarcoma.

Injuries of the Sclera consist of perforations and ruptures. The latter can be caused by blows. Lacking elasticity, wounds of the sclera do not gap unless some tissue, such as the ciliary body or choroid, is forced into the wound. Where the wound extends into the cornea the iris will likely prolapse into it. Infection is to be expected unless the conjunctiva is uninjured.

Infection is followed by uveitis or panophthalmitis; the globe is prone to soften and shrink. The fellow eye may be attacked by sympathetic ophthalmitis later. Bandage to the eye and rest in bed are indicated. Sutures are seldom needed, but large wounds may require them. A badly lacerated eyeball without light perception should be enucleated at once.

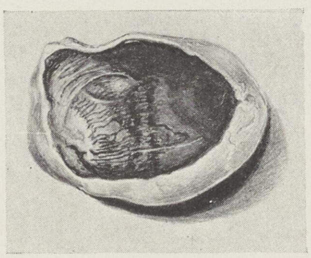


Fig. 120.

Staphyloma or ectasia of the sclera arises when the scleral wall is too weak to resist intra-ocular pressure. It is usually localized. The general form is found only in children; this is illustrated in buphthalmos, in which the cornea also is notably expanded. When the posterior part of the sclera stretches a staphyloma posticum is formed and myopia results. Localized staphylomata may be single or multiple; where they occur over the ciliary body they are called anterior, but when they arise from the interval between the ciliary body and the iris they are "intercalary." The multiple form may become confluent.

A staphyloma is recognized as a noninflammatory bluish elevation; the color is derived from underlying uveal tissues. The elevation can be transilluminated from the pupil, or the pupil from a light in contact with the elevation. The prominence may interfere with the closure of the lids (Fig. 120).

Treatment is directed toward the underlying condition. Miotics or an iridectomy are indicated to lower glaucomatous tension. When the globe is painful, or the lid cannot be closed, or the eye is unsightly, enucleation may be advised; there is no possibility of restoring form or function.

Where the eyeball protrudes the lids may be stretched considerably, so that after enucleation an artificial eye is retained with difficulty. No plastic shorten-

ing of the lid should be done until at least one year has elapsed after removal of the globe; by that time the lid usually accommodates itself to the prosthesis. The stretching of the upper lid is accompanied by a persistent recession of the soft tissues beneath the superior margin of the orbit. Plastic operations to fill the depression are not recommended.

Blue Sclerotics.—In blue sclerotics the white of the eye has an azure or a china blue appearance, because of an undue translucency or a uniform thinness, not stretching, of the sclera, which allows the underlying uveal tissues to show through. In some cases the width of the ciliary body is plainly outlined. Blue scleræ may appear spontaneously, or as a dominant hereditary characteristic which can be transmitted by either affected parent.

The association of blue scleræ and weakness of fibrous structures throughout the body have been noted. The abnormalities are due to defects in the development of tissues which are derived from mesenchyme. Hypotonicity or laxity of joint ligaments and thin-walled long bones contribute to the occurrence of sprains, dislocations and fractures from insignificant causes.

Blue scleræ are essential to the diagnosis of true hereditary fragilitas ossium. The remaining symptoms are hypermobility of joints and hereditary otosclerosis or nerve deafness; occasionally there is a history of fractures from trivial injuries. Fractures are commonly limited to the period of childhood, and deafness occurs before reaching maturity. Embryotoxon has been observed in some of these cases. The patient is usually of short stature and small frame. Hysterical and emotional manifestations are not uncommon.

The presence of associated symptoms and a positive history, and the absence of buphthalmos and axial myopia will differentiate "blue sclerotics and brittle bones" from other conditions. There is no treatment.

CHAPTER VII

THE OPHTHALMOSCOPE AND OPHTHALMOSCOPY

The ophthalmoscope is valuable in routine physical examinations because the transparent media of the eye permit a direct inspection of living tissues. In general diagnosis the clinician reasons largely by deduction, and relies chiefly on the lessons of pathology for the explanation and interpretation of morbid conditions. Because of the accessibility of the eye, the state of ocular structures and tissues can be determined with an accuracy not possible in any other organ. These tissues frequently exhibit evidence of the character of a systemic disease.

By means of the ophthalmoscope the examiner can bring into focus and magnification the vitreous, retina, choroid and nerve head. The discovery of an intra-ocular lesion, and a study of its site, features and progress afford conclusions not available by any other means. Fine points of diagnostic importance can often be seen with the instrument when they cannot be made out at all or only with difficulty otherwise, or perhaps until the disease has progressed to full development. Diagnosis, treatment and prognosis are influenced by the presence or absence of the signs of structural changes.

Ophthalmoscopy is an adjunct to other methods of examination; it should amplify and not take precedence over them, and an undue importance must not be accorded to it. Experience is required to form an adequate judgment of what is seen; practice brings the experience, and with it confidence in an ability to detect early changes at a time when their recognition affords an opportunity for more effectual treatment and a better prognosis.

The application of ophthalmoscopy is included in various sections of this text, and it is especially emphasized in the examination of the vitreous, choroid, retina and optic disk, and in glaucoma. Familiarity with the normal fundus is essential to the recognition of abnormal conditions. Proficiency comes only from routine practice, or the habit of looking into the fundi of every patient at every opportunity.

Ophthalmoscopes are designed to illuminate the interior of the eye without reflecting the source of light into the eye of the surgeon. They are of two kinds. The reflecting instrument depends upon an outside source for light that is reflected into the eye by a mirror; the simplest device is a hand retino-

scope. The electrically lighted instruments illuminate from a self-contained bulb direct to the eye, or by reflection from a mirror in the head of the instrument. The current is supplied by a battery in the handle or from a resistance lamp. The electrical instrument can be used for bedside examinations.

Several ophthalmoscopes are on the market; they are all constructed on the same principle, in that each carries a battery of spherical lenses which generally range from +32 diopters to —36 diopters. These lenses can be rotated before the aperture or sight hole of the instrument by means of a wheel with a milled edge. In using the instrument, the surgeon should keep his forefinger on the milled wheel to facilitate changing the lenses.

There are three methods of practicing ophthalmoscopy. For the *direct* method the surgeon takes the instrument in his right hand, and with his left thumb holds the upper lid of the patient's right eye open. The position is reversed for the left eye. The patient is directed to look steadily at a distant object so that his eye will be fixed in position. The surgeon's and patient's heads should be on the same horizontal plane. The surgeon then sights through the aperture of the instrument at the patient's pupil from a distance of 15 to 30 centimeters (6 to 12 inches), and about 15 degrees temporally from the patient's line of sight. In this position the white reflex of the optic disk will be recognized.

The surgeon then approaches the patient, keeping the disk in view, until the aperture is a half inch or so from the patient's eye. By changing the lenses with the finger on the wheel, one will be found which gives the clearest view of the fine vessels on the disk. An alternate method of finding the disk is to locate a retinal vessel, and then follow it in the direction toward which its size increases.

Since the surgeon is looking at an object very near his eye, he will unconsciously exercise his accommodation; he must needs use a corresponding concave lens in the aperture to neutralize his accommodation, unless the patient is hyperopic to the same amount. The better plan is to keep both eyes open and try to see the fundus as though it were a considerable distance away. When the experiment is successful, the accommodation will relax.

The corneal reflex will annoy; unless the pupil is very small, it is usually possible to sight between the reflex and one border of the pupil, or change the direction of the light and thus displace the reflex. By these means a very good view may be had of the disk and adjoining structures.

For the *indirect* method the surgeon holds the instrument as before. With his left forefinger and thumb he holds a convex sphere of 13 D. 7.5 centimeters (3 inches) in front of the patient's left eye; the tips of the ring and little fingers rest lightly on the forehead, and the arm is in such a position that it

will not obstruct the patient's line of sight. The patient is directed to look past the surgeon's left ear at a distant object. The position is reversed for the right eye.

When the lens is held in the proper position and at the correct distance, an inverted image of the disk can be seen about 7.5 centimeters (3 inches) in front of the lens, provided the surgeon is not too close to the patient; he should approach from a distance of about 45 centimeters (18 inches). One must fix on this position in front of the lens in order to see the image.

By moving the lens about before the observed eye, and nearer to and farther from it, and by varying the distance between the observing and observed eyes, the image can be brought into sharp focus. Adjoining structures are brought into view by tilting the lens, or by moving it about, or by the surgeon moving his head about. The image moves in the same direction the lens is moved, and opposite to the direction that the surgeon moves his head. By observing these rules, the periphery as well as the posterior eye-grounds may be seen. The corneal reflex can be displaced by tilting the lens or by changing the direction of the light.

By either method the macula can be seen by directing the patient to look into the aperture of the instrument or at the mirror. This usually causes him to quickly close his eyes. To see the macula without especial discomfort to the patient, the surgeon can fix on the disk, and then sweep the light across the fundus temporally. A glimpse of the disk may be had in passing. For more prolonged observation, the macula may be studied in the twilight zone that surrounds the brightly illuminated area, or the light may be reduced.

By the direct method the surgeon sees an upright image of the fundus magnified about fourteen times; by the indirect, an inverted image magnified about four and one-half times. The direct method affords a view of a small area of the fundus; by moving the instrument, successive areas are illuminated. The indirect method allows a view of a larger area at once, and a little sharper outline of the disk. Where the nerve looks swollen or blurred by the direct method, it should be inspected by the indirect; in pseudoneuritis the disk margin can be made out, although it will still appear blurred in optic neuritis and in papilledema (see Plate X, Figs. B and C). Otherwise the direct method affords the better detail. It is possible to measure differences in depth by the direct method only.

The *direct-indirect* method consists of reflecting a light into either eye with a concave mirror from distances of 75 to 15 centimeters (30 to 6 inches). The patient is directed to move his eye about. Gross conditions are quickly seen by this method.

Retinal reflexes are troublesome when the light is too bright, and in younger

patients the "shot-silk" retina may give the impression of pathology that is not present. Retinal reflexes are not fixed in position, while lesions are. It is usually better to use low illumination. Fine details are usually seen more distinctly in the twilight zone that surrounds the lighted area, since they are often invisible under very bright light. The alternative is to hold a piece of daylight glass before the patient's eye. The retina can be studied best with a red-free filter; the choroid is not seen through it.

It is always desirable and sometimes necessary to dilate the pupil. The surgeon should first make an inspection to see that no retinal vessels drop abruptly over the margin of the disk; he should then take the tension with an approved tonometer. When both examinations are negative, a drop or two of 2 per cent solution of homatropin usually provides sufficient dilatation for an eye-ground examination. After this has been made, a drop of 1 per cent solution of pilocarpin should be instilled in each eye.

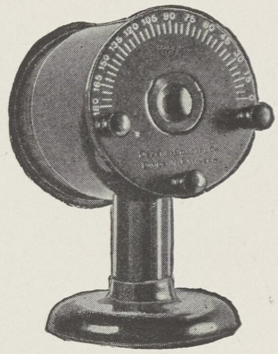


Fig. 121.

The anterior surface of the eye can be inspected with a plus 20, and the pupil and iris with a plus 16. Opacities in the lens can be seen with a plus 12 to plus 7. Fixed and floating opacities in the anterior vitreous are visible with a plus 7, and those in the posterior with a plus 4. The retina, choroid and nerve head are usually seen with the aperture or a weak plus or minus lens.

Profitable practice is available with a schematic eye (Fig. 121). The background is painted to represent a fundus. The glass window in front is a lens that represents the cornea. By adjusting the telescopic slide in the back of the instrument, the eye may be made myopic, emmetropic or hyperopic. Slots before the pupil are provided for holding spheres or cylinders or combinations of them from the trial case. The axis marks indicate the positions of cylinders. The eye is conveniently planned for the study of ophthalmoscopy and retinoscopy.

CHAPTER VIII

VISUAL FIELDS AND BLIND-SPOTS

THE FIELD OF VISION

The binocular field of vision refers to the space in which movements of objects may be detected by the eyes while they are in the primary position. The size of this space depends upon several factors: the eyes may be prominent or deeply set, the nose may be large or small, and the brows may overhang or not.

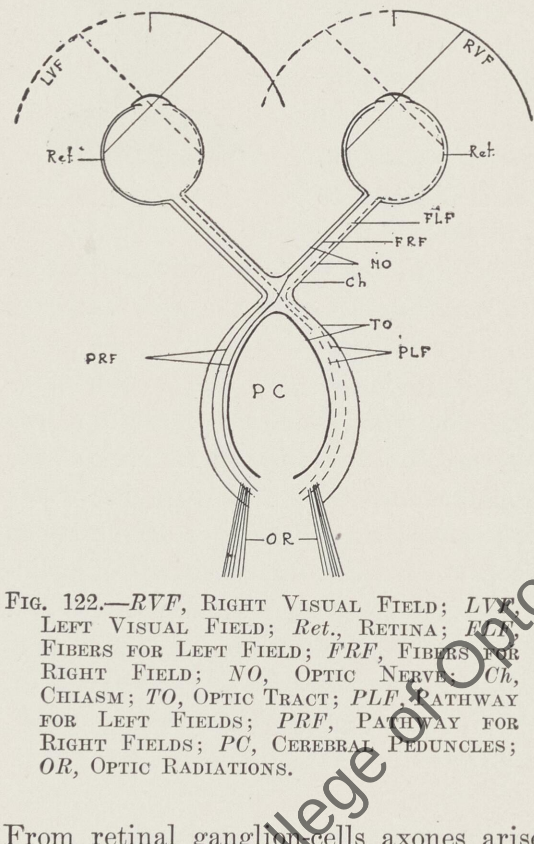


Fig. 122.—*RVF*, Right Visual Field; *LVF*, Left Visual Field; *Ret.*, Retina; *FLF*, Fibers for Left Field; *FRF*, Fibers for Right Field; *NO*, Optic Nerve; *Ch*, Chiasm; *TO*, Optic Tract; *PLF*, Pathway for Left Fields; *PRF*, Pathway for Right Fields; *PC*, Cerebral Peduncles; *OR*, Optic Radiations.

By the diagram (Fig. 122) it is shown that light rays from objects must pass through the pupil; when they come from the left side, they pass to the right half of the retina; when they come from below they pass to the superior half, and when from the upper field they pass to the inferior half. From a compilation of figures, the following dimensions in degrees are given for the average field: Temporal 90, superior 48, nasal 55, and inferior 65. The average fields for blue, red and green are shown in Figure 123. These are ordinarily about 10 degrees apart from without inward in the order named.

Each retina is divided vertically through the fovea into a temporal and a nasal half for clinical study. From retinal ganglion-cells axones arise and pass backward along the optic nerve to the chiasm. Here the axones from the temporal half of the retina continue into the optic tract on their own side, while those from the nasal half cross to the optic tract of the opposite side, and thereby decussate with axones from the nasal half of the opposite retina. From the tract the visual

pathway for each side goes to the visual area about the calcarine fissure in the occipital lobe of the corresponding cerebral hemisphere; consequently, the right half of *each* retina is represented in the right lobe, and the left half is represented in the left lobe.

A total lesion of the right optic nerve results in total blindness of the right

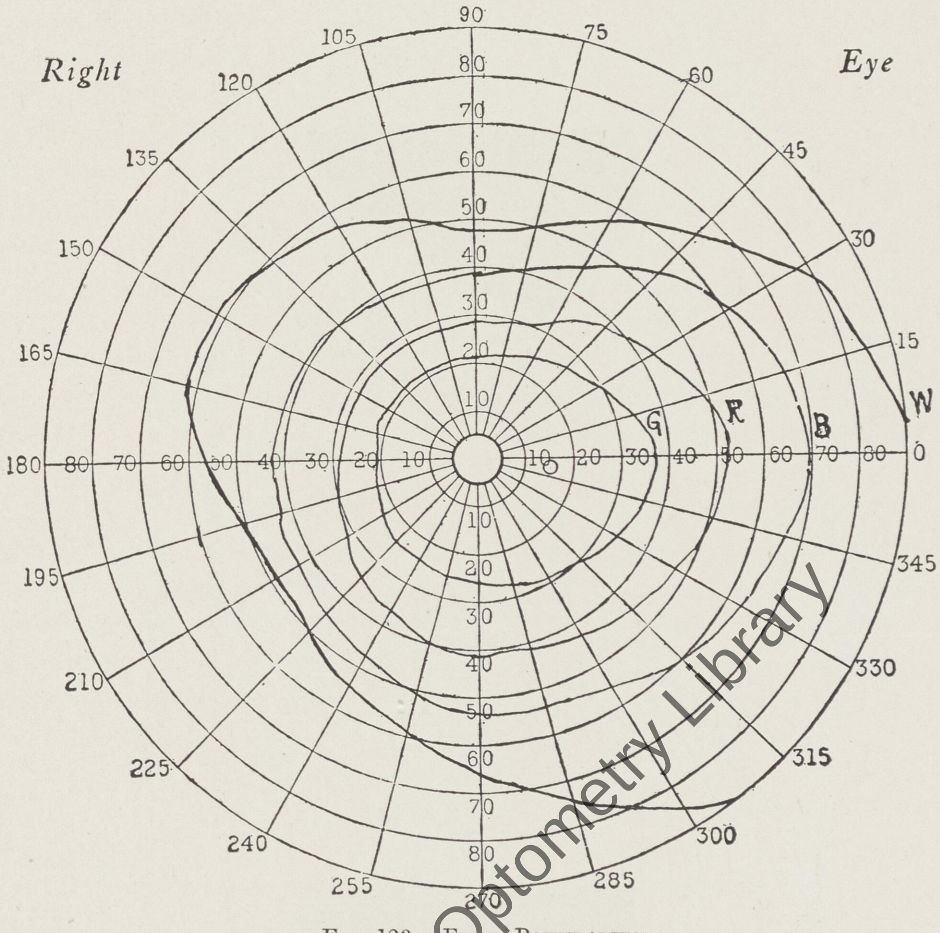


Fig. 123.—Field Boundaries.
W, white; *B*, blue; *R*, red; *G*, green.

eye. A lesion that involves the temporal half of the nerve only will result in a loss of the nasal field; one that involves the nasal half will cause a loss of the temporal field. A lesion of the superior half of the nerve will result in a loss of the inferior field, and a lesion of the inferior half will cause a loss of the superior field. A lesion of any corresponding half of the retina will result in losses comparable to those caused by lesions of the nerve. Partial losses may occur in any part of a field.

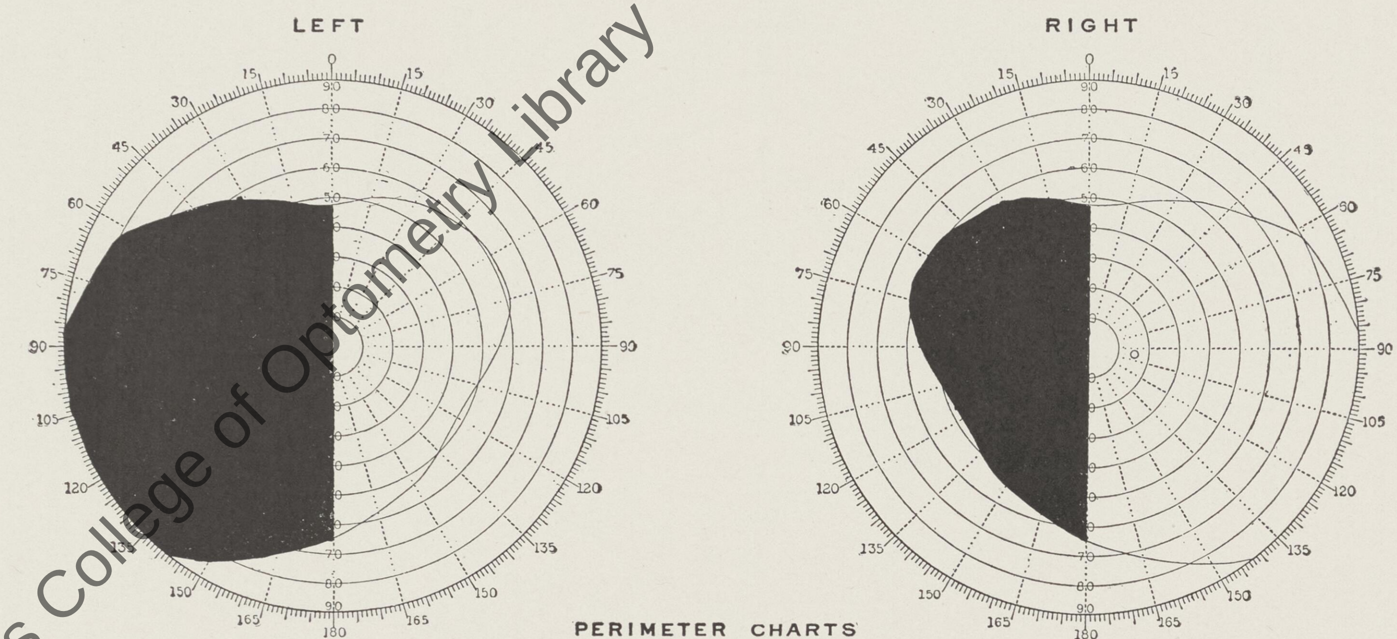


FIG. 124.—LEFT HOMONYMOUS HEMIANOPSIA.

A total lesion of the right optic tract, or of any part of the path posterior to the chiasm, will result in the loss of the left field for both eyes. This is called homonymous hemianopsia (Fig. 124). An incomplete lesion behind the chiasm will cause a partial loss in a corresponding part of the left field of each eye. Partial losses are called quadrant and sector anopsias, according to the amount of field involved; they can occur in one or both fields.

It has been observed that the central area of the field of each eye is preserved in some cases of homonymous hemianopsia; this indicates an intact papillomacular bundle in the visual pathway, and an intact macula. Various theories have been advanced to account for this. Central vision is believed to be represented in the posterior pole of the occipital cortex. This part is supplied by branches of both the middle and posterior cerebral arteries. When one vessel is incompetent, the other remains to supply the part. As long as the central visual area is nourished and the conduction fibers and macula are intact, central vision will be retained.

In transient right homonymous hemianopsia such as sometimes accompanies attacks of migraine, the patient has central vision, but experiences difficulty in reading print from left to right; there is no difficulty in reading Hebrew or numerals from right to left.

Lesions of the chiasm result in total or partial binasal or bitemporal hemianopsia. An anteroposterior division of the chiasm divides that structure into lateral halves; thus it interrupts the continuity of the decussating fibers. Since these fibers convey visual impulses from the nasal halves of both retinæ, it follows that the temporal fields will be dark (Fig. 125). When the lesion invades the superior half of the chiasm, the inferior halves of the fields are dark: when it involves the inferior half, the superior halves are dark. A lesion may be circumscribed and affect the field of one eye only.

Tumors and cysts of the pituitary body are usually accompanied by bitemporal hemianopsia, but not invariably so. Enlargements of the gland cannot be excluded because of the absence of the characteristic fields. A roentgenograph of the sella turcica should be made in suspected cases. During pregnancy there is ordinarily some limitation of the temporal fields, because of the physiological hypertrophy of the gland. Distentions of the third ventricle can encroach on the chiasm.

A lesion in one or both lateral angles of the chiasm can destroy the fibers from the temporal half or halves of the retinæ. In such cases the hemianopsia is nasal or binasal. For the production of the very rare binasal type, a bilateral lesion is required. Aneurysms of both internal carotid arteries at this location could cause it.

Lesions which affect the anterior portion of the retina will contract the

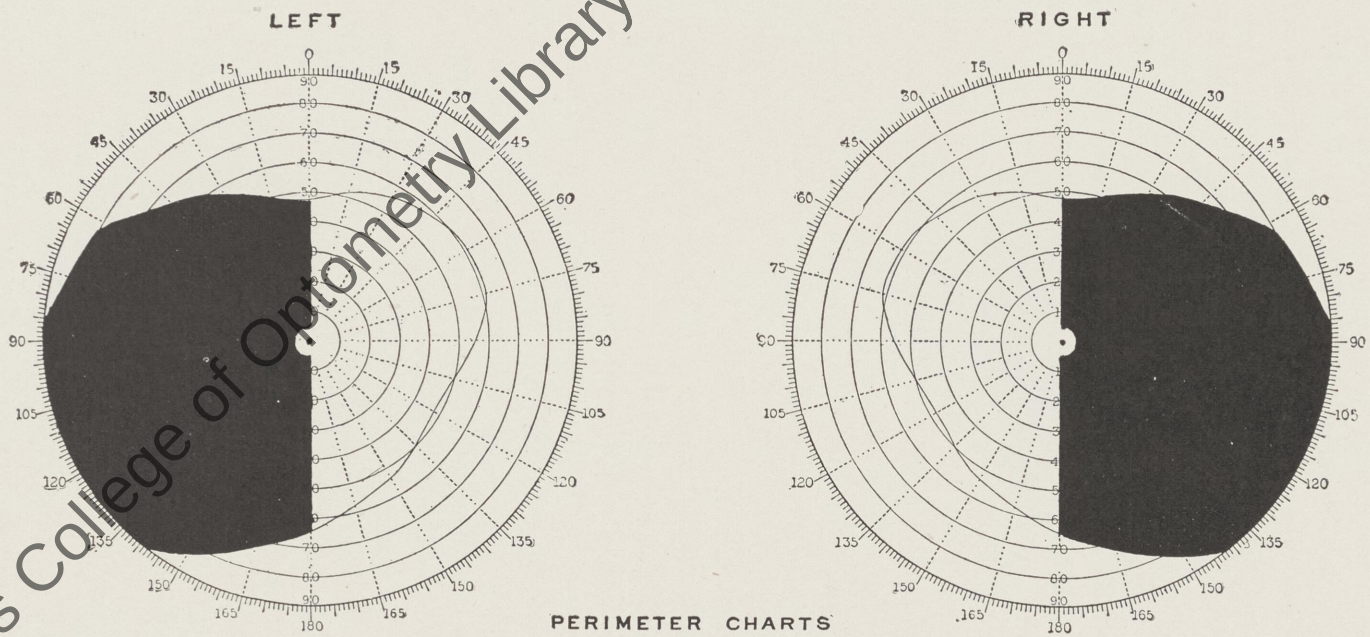


FIG. 125.—BITEMPORAL HEMIANOPSIA.

visual field concentrically; that is, the entire field will be restricted. The condition can be bilateral.

The boundaries of the visual fields are charted by the aid of an instrument called the perimeter. This consists of a metal semicircle or perimeter arm suitably mounted for use. At the center of the arm is a fixation device. The arm is marked off at intervals of 10 degrees. Schweigger's hand perimeter is useful for all ordinary purposes (Fig. 126); it is small and portable. The fixation device is a small mirror in which the patient sights on the image of his eye. The arm is 150 millimeters distant, and the image is twice that. Continuous with the handle is an upright arm which supports a block to fit against the lower margin of the orbit.

The test objects used with this instrument are 2 millimeter and 5 millimeter round disks made of white and of "saturated" colors, blue, red and green, which are mounted on slender rods. The record should note in millimeters the size of the object and the distance at which it was used. This is written in the form of a fraction, the numerator of which indicates the diameter of the object, and the denominator the distance. In this instance the fraction is either 2/150 or 5/150; the visual angle can be calculated from the fraction. The record also should specify the comparative or relative intensity of light used to illuminate the objects. Daylight is usually considered better than artificial lighting.

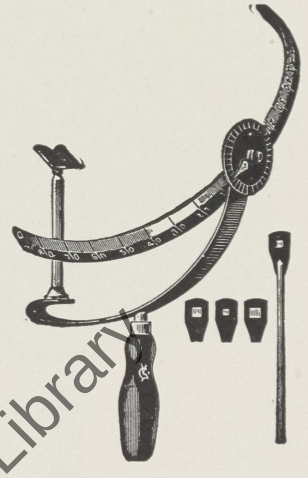


Fig. 126.

The patient is seated so that the light falls evenly on the surface of the arm facing him. One eye is occluded. The patient then sights the image of his other eye in the mirror. A test object is carried from without inward along the arm of the perimeter until it is perceived and its color named. The degree mark on the scale is noted and recorded, as it is for each observation. Eight positions are taken: temporal and nasal, from above and from below, and for each of the four diagonal directions. For white and colors inclusive, thirty-two readings are taken for each eye. These are recorded on a chart, and the points for each color are connected by a continuous line (see Fig. 123).

The examiner must have due regard for the patient and his physical condition. Attention, fixation, and visual perception are readily fatigued. The examination must not be hastened, or the fields will appear smaller than they should and will be misleading. The patient should close the eyes while the points are being marked on the chart.

BLIND-SPOTS

A scotoma is a blind or an indistinct spot or area in the field of vision. The classical scotoma is the blind-spot of Mariotte; it is projected into the visual field from the optic disk. In the average eye the center of the blind-spot is about 15.5 degrees from the point of fixation, and about 1.75 degrees below a horizontal line drawn through it. This scotoma measures about 5.75 degrees horizontally and about 7.5 degrees vertically.

The location and extent of the blind-spot is plotted on a screen or with a campimeter. Peter's campimeter (Fig. 127) is the simplest effective device for this work. The surface is a neutral gray; the point of fixation, lines which radiate from it, circles indicating distances from it, and Mariotte's blind-spot are marked with inconspicuous lines. For refined work a tangent curtain is placed at 750 or 1,500 millimeters from the patient, and very small test objects are used to detect the earliest changes.

Fig. 127.

With the campimeter at close range, 150 millimeters, the objects form larger visual angles than they do at greater distances (see Fig. 283). The size of the visual angle determines the number of retinal elements stimulated by the test object. The advantages of the nearer distance are that the patient can more easily concentrate on the point of fixation, and that he obtains a sharper definition of the objects and recognizes them more promptly, especially the colored ones. The objects vary in size from 1.5 to 20 millimeters in diameter; their size and the distance at which they are used are written in the form of a fraction.

The patient is seated as for an examination with a perimeter, and one eye is occluded. The instrument is held so that the plane of its surface is perpendicular to the line of vision; the gaze must be steadily maintained toward the point of fixation. A small object is moved slowly from within the blind-spot area until its color is recognized; this is repeated in several directions and the readings are recorded on a special chart (Fig. 128). The white, blue, red and green blind areas are outlined. This gives the size of Mariotte's blind-spot.

An enlargement of 1 degree creates suspicion, an enlargement of 2 degrees generally means disease, and an enlargement of more than 2 degrees certainly

means that disease exists (see Plate X, Fig. B). An exception is the enlargement due to the presence of circumpapillary medullated nerve-fibers; these are usually irregular in outline, and they are without pathological significance.

The test objects are next moved from without inward in a radial manner from several positions on each border of the screen; they can also be moved from the fixation point outward. They should be moved spirally, and also parallel with the circles which indicate the various distances in degrees from the center. Should the object become indistinct or disappear, a scotoma exists. Its extent can be outlined in the same way that the area of the normal blind-spot is measured. A permanent record should be made at each examination.

Pathological scotomata are classified variously. A *positive* scotoma is recognized by the patient as a dark area in the visual field; a *negative* scotoma is only discovered by examination for it. In an *absolute* scotoma neither form nor colors are recognized; in a *relative* scotoma, form is recognized but colors are indistinct or invisible.

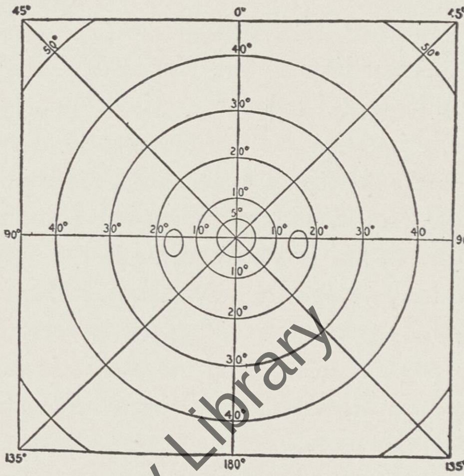


Fig. 128.—Campimeter Chart.

According to location in the visual field, scotomata are identified as *central* when the fixation point lies in the center of a dark spot 5 degrees in diameter. A *pericentral* scotoma curves around the point of fixation but does not include it. A *paracentral* scotoma lies alongside the point of fixation but not beyond 8 degrees nasally and 12 degrees temporally. An *annular* or *ring* scotoma lies between 15 degrees and 30 degrees from the fixation point; Seidel's (see Fig. 163) and Bjerrum's (see Fig. 164) signs as found in glaucoma are examples of this type, and are called *cecal.* Scotomata are *intermediate* when they are located between 25 degrees and 60 degrees from the fixation point, and *peripheral* when they are beyond 60 degrees.

The fixation point corresponds to the fovea centralis. Macular degeneration and lesions of the papillomacular bundle in the optic nerve obliterate central vision and produce central scotomata. This type is usually bilateral; they are difficult to demonstrate by monocular fixation, because extrafoveal vision is serviceable for all purposes except fine work. These cases can be examined by binocular fixation; neither eye is occluded.

A red glass is held before one eye while the other is being tested. To an eye behind a red glass a red test object appears white against a white back-

ground, and a blue object appears black against a black background; the colors are recognized without confusion by the eye not covered with a red glass.

Lesions in the retina or choroid cause scotomata in the field corresponding to their situation. Lesions in the visual paths, including the lateral geniculate body, the pulvinar and the visual cortex produce dark areas in the fields according to the location of the lesion and the amount of destruction done. When these are found, their extent is ascertained in the same way that the blind-spot of Mariotte is outlined.

In general, losses of perception for red and green denote impairment of conductivity or nerve disease, while a loss of perception for blue indicates injury to the percipient elements from disease of the retina or choroid.

COLOR VISION

According to the Young-Helmholtz theory of color vision there are three fundamental color sensations, one each for red, green and violet. All other color impressions are the results of multiple stimulation. No theory yet satisfies all questions concerning color perception, but this one is largely accepted.

Color-blindness is graded according to degrees. An individual may be blind to red or to green, or to red and green; he may be blind to blue also, and he may be totally color-blind. Many tests have been devised. That of Holmgren is perhaps most generally known and employed; it is one of the easiest to pass, and for that reason it is not accepted by discriminating examiners. Special lanterns have been constructed for testing the color vision of railway and marine employees. These lanterns can be manipulated to imitate the various lights as they might appear under a wide range of atmospheric conditions. The doubtful case may be one of simple color ignorance.

A simple test apparatus consists of an oblong box divided into two main compartments, and a number of red, yellow, green and blue beads. One compartment is lined with white and contains the beads. The second compartment is subdivided into four small ones, each of which is provided with a cover so arranged that the examinee cannot see the contents. The test consists of depositing the beads of each color into separate compartments.

CHAPTER IX

THE UVEAL TRACT

The uveal tract consists of the iris, ciliary body and choroid. It is the vascular coat of the eyeball, and its vessels supply nutrient fluids to all the structures inside it except certain layers of the retina. It is deeply pigmented in the normal eye. Compared to a camera the choroid is the dark lining, the ciliary body is the focusing device, the iris is the diaphragm and the pupil is the aperture in front of the lens. Uveal structures are of mesodermal origin and are subject to the diseases which attack such tissues. (See Intrinsic Muscles.)

THE IRIS

Essentials of the Anatomy.—The iris is a diaphragm suspended between the cornea and the crystalline lens. It arises from fibers derived from the pectinate ligament and the ciliary body. Its free border surrounds the pupil. The anterior layers of the iris are composed of modified fibrous tissue which supports blood-vessels, branched pigmented cells and smooth muscle-fibers. The muscle-fibers are arranged circularly around the pupil.

The anterior surface is covered with endothelium continuous with that of Descemet's membrane. In this surface are openings or crypts by means of which aqueous fluid has access to the spongy iris stroma. The crypts are not lined with endothelium.

The posterior layers of the iris are made up of retinal tissue containing pigment and radially disposed smooth muscle-fibers which are apparently of epithelial origin. These fibers dilate the pupil. The retinal pigment is rather constant, while the iris pigment varies. The latter is scant or absent at birth, and the former shows through intervening tissues as blue or gray. As iris pigment forms the color deepens; the color of an iris depends upon the amount of this pigment. Neither pigment is present in pure albinos. The retinal pigment can be seen as a dark rim around the pupil.

The free border of the iris is in contact with the lens. Adhesions between them are prevented during health by the aqueous and the incessant and rapid changes in the diameter of the pupil. When the iris lacks the support of the lens from dislocations or absence of the latter, it trembles, iridodonesis, with every movement of the eye or head, and the anterior chamber is deep.

The iris is rich in blood-vessels. An arterial plexus surrounds the zone of circular muscle-fibers; it anastomoses with another plexus near the periphery by means of radiating arteries. The blood supply is from the anterior and posterior ciliary branches of the ophthalmic. Because of the rich vascularity, congestions occur readily. The nerve supply is sensory by way of the ciliary ganglion from the trigeminus, pupillary contracting by way of the same ganglion from the motor oculi, and pupillary dilating from the sympathetic by way of the anterior ciliary nerves.

In a blue or gray iris slightly elevated wavy radial lines can be traced from near the pupil to the periphery; they are less easily seen in a dark iris. The crypts are more readily demonstrated in light-colored eyes. Patches of brownish pigment often give the iris a spotted appearance. These several features constitute the pattern. Familiarity with the normal pattern is essential to the recognition of iris disease.

Diseases of the Iris

The iris should be examined by focal illumination and magnification from different angles in order to exclude changes in the cornea and aqueous. The patient can be instructed to rotate the eye in different directions, or the examiner can vary his own position. Both eyes should be scrutinized, for by comparison objective evidence of monocular disease is obtained. Occasionally the irides of an individual are unlike in color, heterochromia, but this seldom has any pathological significance. The acquired form is seen where one iris is atrophied from iritis or glaucoma.

Hyperemia of the iris develops from the presence of a retained foreign body on the cornea or similar irritation, or it is the first stage of an iritis. It is caused by engorgement of the iris vessels. The effect is to thicken the iris, contract the pupil and limit its mobility. The thickening modifies the color; a light iris appears somewhat greenish, while a darker one appears reddish brown. The pattern is obscured, and the iris looks blotched or muddy. The cornea is usually clear enough for the iris to be seen easily.

The pupil is contracted and reacts sluggishly because of the congestion and the irritation to the pupilloconstrictor nerves. Mydriatics act slowly and incompletely, and their effect is of shorter duration than usual.

The engorgement extends to the deep circumcorneal vessels, which radiate from the cornea for a distance of 3 to 4 millimeters as violet striations. The conjunctival vessels are moderately engorged and bright red. They form an anastomosing network which does not extend as far as the cornea.

The symptoms are pain, photophobia, increased lacrimation and blurred

vision. The treatment is to dilate the pupil and protect the eye from light by dark glasses.

Iritis is primary when the iris is the first ocular structure involved, and secondary when it follows a disease or injury of another structure. According to its course, it is acute or chronic. Consideration of signs and the theory of their production are of greater value than symptoms in diagnosis.

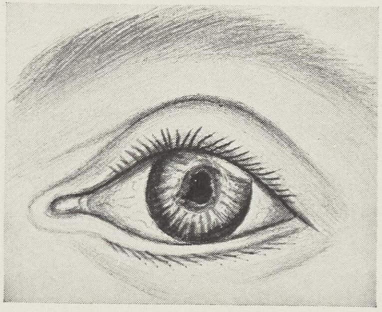


Fig. 129.—Iritis.

Inflammation progresses from a hyperemia. There is first a round-celled infiltration into the iris tissues, and later an exudation into the aqueous chambers. The pattern is further obscured by additional thickening of the structure, by the deposit of the exudate on the surface, and by the presence of suspended clumps of cells in the aqueous (Fig. 129). The cells are deposited upon the posterior surface of the cornea and diminish its transparency (see Fig. 111). Cells settle into the most dependent part of the anterior chamber and hypopyon (see Fig. 107) is formed; when red blood-cells have been extravasated, the hypopyon is reddish and is designated hyphemia.

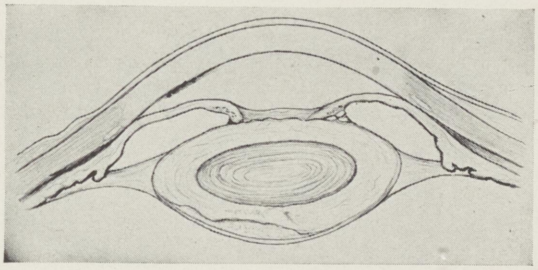


Fig. 130.—Occlusio Pupillæ. (After Fuchs.)

The exudate gradually extends across the pupil and adheres to the capsule of the lens. There is a tendency toward organization when fibrin is present. A pseudomembrane forms across the pupil in contact with the lens epithelium (Fig. 130) but without particular injury to it. As organization progresses the membrane contracts and tears loose from the pupillary border; the free edge curls forward and exposes a ring of black pupil (Fig. 131) surrounding the membranous disk. Where the pupil has thus far failed to dilate adequately to atropin, it may do so now. The process continues until the false membrane has been absorbed or thrown off.

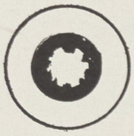


Fig. 131.

When enough fibrin is present the membrane may become so attenuated that it is almost or quite invisible to inspection, but it remains attached and covers or blocks the pupil; this is called "occlusio pupillæ." The eye is permanently damaged.

The excess of fibrin sometimes fills the anterior chamber. It obscures the iris pattern and sometimes completely conceals the iris. In resolution it

shrinks somewhat like a drying sponge; its behavior suggested the name of *spongy iritis* (Fig. 132).

The presence of agglutinating materials in the exudate causes most of the sequelæ arising from the contact of the iris with the lens. When the case is seen in an early stage the pupil will respond to a mydriatic. Examination often discloses a ring of dark pigment granules on the lens surface; this ring corresponds to the original position of the pupillary margin and marks the location of posterior synechiæ when the adhesion of iris and lens is allowed to develop.

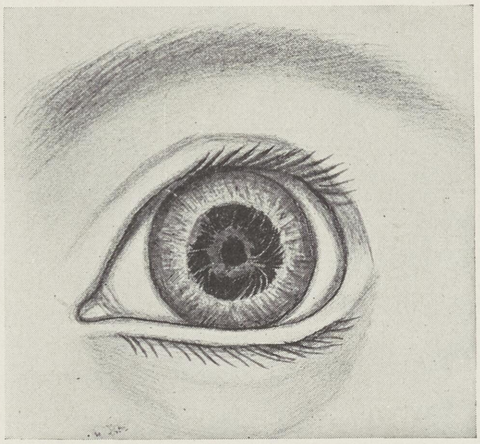


Fig. 132.—Spongy Iritis.

In untreated cases the iris adheres to the lens at one or more points or arcs around the border of the pupil. Should these adhesions break under mydriasis, black dots or crescents of pigment will be found on the lens capsule corresponding in location to the ring described above. When the adhesions hold under mydriasis the pupil dilates unevenly; it is fixed at the adhesions and dilated between them. This accounts for the "clover-leaf" or "ace of clubs" form of pupil (Fig. 133).

Where the adhesion is continuous for the whole extent of the pupillary border the aqueous cannot enter the anterior chamber; this is called "seclusio pupillæ" (Fig. 134). This variety of adhesion is also called *ring, annular, circular* or *complete posterior* synechiæ. The border of the pupil is black from pigment or gray from exudate.

The continued secretion of aqueous presses the iris forward like a filled sail between its periphery and the adherent pupillary margin; this is "iris bombé." When the iris is flat and the pupil is fixed to light, it may be thought that an annular synechia exists; a potent mydriatic will demonstrate the presence of a horseshoe or D-shaped dilatation. The aqueous passes into the anterior chamber through this opening and there is no iris bombé. Where no aqueous is secreted iris bombé cannot form. A fixed pupil must be studied with a mydriatic.

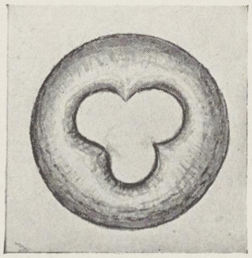


Fig. 133.—Synechiæ.

Exudate in the posterior chamber may seal the iris flatly against the lens and form a *total posterior* synechia (Fig. 135). A ciliary body covered with exudate does not secrete aqueous. *Partial* total synechia exists where parts of

the iris are sealed flatly to the lens while other parts lie forward in folds toward the cornea. The unadherent portions indicate the pressure of aqueous from behind. An iridectomy must be directed into one of these folds, and not into an adherent part. Should any fold be crowded against the cornea it may adhere there and form an anterior synechia. When this occurs an adherent leukoma may develop.

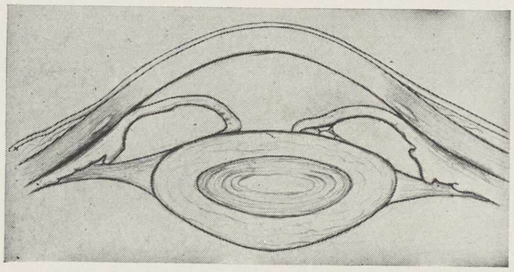


Fig. 134.—Seclusio Pupillæ and Iris Bombé.

An unusual form of posterior synechia occurs while the pupil is dilated, and the adhesion forms near the periphery of the lens. When the pupil contracts the iris folds upon itself; a part of its anterior surface faces the anterior surface of the lens. An angle is formed in the circular zone next the pupil, showing how it is rolled backward or tucked behind the radial zone.

The exudates so far described are organizable or plastic; for this reason the term "plastic iritis" is often used. It is an acute form of inflammation which is subject to recurrences and a liability to become chronic. Where connective tissue replaces iris tissue in the process of resolution the iris undergoes more or less atrophy.

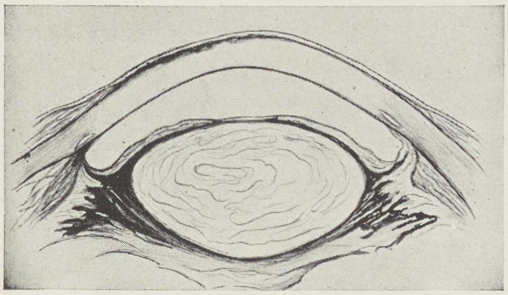


Fig. 135.—Total Posterior Synechia. (After Fuchs.)

Because of the continuity of the iris and the ciliary body, the similarity of their tissues and their common blood supply, an inflammation of the one is generally complicated by an inflammation of the other. While the iris is accessible to inspection, the ciliary body is not. Inflammation of the ciliary body or cyclitis exists where there is localized redness in the sclera at one or more points over the ciliary body, or when tenderness can be elicited by light palpation with the finger tip in this region.

THE CILIARY BODY

Essentials of the Anatomy.—The ciliary body lies against the sclera and occupies a zone 5 to 7 millimeters wide behind the iris. It is attached to the pectinate ligament, the periphery of the iris and the sclera anteriorly, and blends into the choroid posteriorly. It consists of two major portions: the pars

plicata or corona ciliaris, the folded part behind the iris, and the pars plana or orbiculus ciliaris, the flat part that extends to the choroid. The folded portion is triangular in shape on cross section; it contains muscle-fibers, pigment, blood-vessels and nerves. The ciliary processes are continuous with it. The inner surface is composed of two layers of retinal cells similar to those on the posterior surface of the iris. The *ciliary processes* are directed toward the lens. They number about seventy and are credited with the secretion of the aqueous humor, a modified lymph for the vitreous humor and lens.

Diseases of the Ciliary Body

Iridocyclitis refers to an inflammation that involves both the iris and ciliary body. The ciliary body is inflamed when it is tender at any point, when the exudates are abundant and the vision is markedly diminished, where the signs of inflammation include edema of the lids, and when the tension is below normal. Simple cyclitis is characterized by the presence of deposits on the cornea and in the vitreous, and by the absence of pronounced inflammatory reaction.

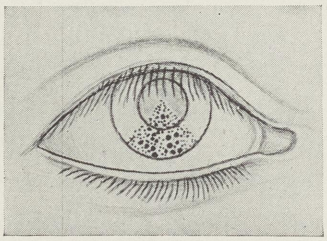


Fig. 136.—Keratic Precipitates. (After Nettleship.)

The formation of hypopyon has been mentioned. Inflammation of the ciliary body and its processes modifies the character of the aqueous because albuminous or proteid materials and fibrin are poured out with it. The exudate gathers in clumps which are more adhesive than accumulations of white cells. They come into contact with the cornea and adhere to its posterior surface. The lighter and smaller ones adhere higher up on the cornea and the heavier and larger ones lower down (Fig. 136). They form as a cone or pyramid whose apex is at or near the center of the cornea. Other clumps become attached at indifferent points (see Fig. 111).

This feature is called punctate keratitis and keratic precipitates or k.p. Former names for the condition were "descemetitis," "aquocapsulitis," "onyx," and serous cyclitis or serous iritis. The last name distinguishes that condition from plastic iritis or iridocyclitis. The term keratic precipitates is descriptive. The clumps are creamy to yellowish in early stages, but they may acquire a brownish appearance later, due to a loss of cytoplasm from the cells without destruction of the nuclei.

Increased tension characterizes one class of iridocyclitis cases. The aqueous contains an excess of albuminous and proteid materials; it is thick or viscid and obstructs the filtration spaces. Aqueous secretion continues and the intra-

ocular tension is raised; the anterior chamber is deep because it is filled with fluid under pressure, the iris is depressed and the pupil is slightly enlarged. This constitutes a secondary glaucoma.

In other cases the secretion of aqueous is diminished. This occurs when the activities of the secreting cells are inhibited by toxins or when the ciliary body is covered with exudate. Decreased tension follows this process and the eyeball softens. The anterior chamber is deep in this group also. The keratic precipitates are seen as fine pale dots on the posterior surface of the cornea; the hypopyon and cone-shaped figure may be absent.

Some of the masses of exudate may be very large and elongated; one end adheres to the cornea while the free end floats in the thickened aqueous. These shreds may be partly composed of endothelial tissue. Destruction of endothelium permits the aqueous to enter the substantia propria, which interferes with its nutrition and clouds it. The epithelium is disturbed too; fluorescein produces a pale stain deep in the cornea.

That the aqueous is abnormal and its secretion diminished is evident from the changes that occur in structures nourished by it. Myopia is occasionally noted in early stages. The lens becomes permanently opaque in late stages. The vitreous is damaged by exudates, toxins or inadequate nourishment. The eyeball remains soft. In time the globe shrinks, and this may be assumed to result from contraction of the vitreous. The patient describes sensations of sparks or flashes of light which are due to localized traction on small areas of the retina, presumably from contracting bands forming in the vitreous. During this stage the iris is retracted at the pupil.

As the inflammation subsides the cornea and aqueous become clear, the iris resumes its accustomed position unless bound by synechiæ, and where the tension has not been raised sufficiently to compress the iris it regains something of its normal appearance. Atrophy of the iris will follow its compression or accompany shrinking of the globe.

Symptoms of iritis and of iridocyclitis are accounted for by the congestion of blood-vessels, the irritation of sensory nerves, and the exudate. They are: Excessive lacrimation, photophobia, pain, blepharospasm and impaired vision. Movements of the iris are painful and light produces a spasm of the sphincter fibers. The pupil normally contracts during sleep, and it is significant that the pain of iritis is usually worse at night. The iris tolerates light better when immobilized by atropin.

Pain may be practically absent or it may be intolerable. Pain in the eyeball is caused by the congestion, but it may radiate to the brow, the nose, the cheek or the teeth. Intense pain may be accompanied by vomiting and constitutional symptoms.

Vision is diminished because of the exudate on the cornea, in the aqueous, blocking the pupil, or in the vitreous.

Etiology.—Iritis is caused by a wide variety of microörganisms. About half of the cases are due to syphilis. Another group of cases is due to foci of infection in the accessory cavities of the nose, the teeth, tonsils, appendix or gall-bladder. Some cases are complications of chronic gonorrhea, gonorrheal prostatitis or chronic seminal vesiculitis, of the acute infectious diseases such as meningitis, pneumonia, or influenza, and rarely it occurs in metabolic disturbances such as diabetes. Nodular iritis due to syphilis, tuberculosis or leprosy is very rare.

Syphilitic Iritis may be diagnosed when the regulation tests are positive, or when antisyphilitic treatment is followed by prompt improvement. The nodular form is discrete, multiple or single, and mostly occurs in the sphincter zone or at the pupillary border. Broad resistant synechiæ form. The nodules are associated with early symptoms and are not regarded as gummata. Participation of the whole iris may be looked for in the secondary stage of syphilis. Iritis develops in both the acquired and congenital types of the disease. Choroiditis and neuroretinitis often accompany the latter form. The treatment is local and constitutional.

Tuberculous Iritis occurs as a solitary tubercle, a very fine miliary form, and a combined exhibition of fine miliary points and various sized nodules. It is seldom found in persons after the twentieth year. Synechiæ form, but the manifestations tend to completely disappear. Absence of pain is significant. Ocular tuberculosis is secondary or metastatic, although the first manifestations of the disease may appear in the eye. Tuberculin should be used for diagnosis and treatment.

Leprosy of the iris is extremely rare, and has been noted mostly, if not entirely, in the form of nodules.

Rheumatic Iritis, so called, is caused by some focal infection, but not usually until after the appearance of other secondary manifestations such as subacute arthritis, endocarditis, or depressed constitutional states. This form is subject to the same recurrences that characterize the antecedents. While the first attack may be followed by apparent complete recovery, subsequent attacks leave permanent and serious changes in the tissues. This form is many times diagnosed when a careful history and a thorough examination would direct attention to the genito-urinary tract.

Gonorrheal Iritis is usually severe and is often limited to one eye. It does not follow an acute gonorrhea, but comes on as a late manifestation of a gonococcal focal infection; for instance, after arthritis, particularly of the knees. It is advisable to look for the infected focus in the seminal vesicles and the

prostate gland. Iritis may accompany a recurrent urethral discharge, in which the prostate gland frequently plays an important part. It resembles the rheumatic form in severity, course, and sequelæ, although, unlike the latter, exposure to cold and wet does not bring on a recurrence. The complement fixation test may be of value.

An iritis which develops in the course of or soon after an acute infectious disease has probably been caused by that disease. The same observation holds in the presence of a recognized metabolic disturbance like diabetes.

While there is little local evidence to distinguish a syphilitic from a non-syphilitic initial attack of iritis, the former leaves more pronounced proof in the form of synechiæ or of pigment spots on the lens capsule. After the attack has subsided, the media may clear sufficiently to permit an ophthalmoscopic examination. Should the choroid, retina and optic disk show characteristic changes, syphilis may be accepted as the cause of the iritis, regardless of negative laboratory findings.

In an analysis of one hundred consecutive cases of iritis, Bulson observed that there was less pain in syphilitic iritis than in other forms, and that pain was especially severe when the teeth or tonsils were at fault. The iris was principally involved in the syphilitic form, while the ciliary body participated actively in other forms. Dental focal infections occurred mostly after forty, and tonsillitic types before that age. The probability of changing a negative blood Wassermann test into a positive one by a provocative intravenous injection of neosalvarsan is emphasized.

UVEITIS AND IRIDOCYCLITIS

Diagnosis of iritis is made principally from the appearance of the eye. The iris is "muddy"; the pattern is not clear. The affected iris may be compared to that of the fellow eye or some unaffected eye. The cloudiness of the cornea or aqueous may make inspection difficult or impossible, for the eye is not seen by the physician until pain or diminishing vision urges the patient to seek advice. A careful inquiry into the past personal history of the patient, and a search for evidences of past or present illness and foci of infection are important.

Where the iris is obscured or hidden, some of the following signs are usually present: Deep pale staining by fluorescein, fine creamy dots or masses of exudate on the posterior surface of the cornea, principally in the lower half, hypopyon that is unaffected by gravity, aqueous cloudy or containing shreds, gray pupil, pupil contracted or irregular in outline, pink or violet wavy or straight vessels radiating from the border of the cornea a short distance, ten-

derness over the same area, and an anterior chamber of normal or increased depth.

Iritis has been confused with glaucoma and with conjunctivitis. To treat a conjunctivitis as one of the other diseases seldom results in serious consequences but is a mistake nevertheless. To treat one of the others as a conjunctivitis is a serious error which cannot be condoned because the eye may be permanently damaged by improper treatment and by delay in receiving proper attention.

To treat glaucoma as iritis, or iritis as glaucoma constitutes one of the gravest mistakes that can occur in the management of eye cases, for the treatment is fundamentally opposite; the pupil must be dilated in iritis, and contracted in glaucoma. While no case will illustrate every sign and symptom, the systematic table of differential diagnosis (page 153) will be found helpful.

Secondary glaucoma due to iritis has been mentioned; the pupil is somewhat dilated, the anterior chamber is deep, and the tension is raised. In such cases the iritis will probably be recognized as the dominant or primary disease. An acute glaucoma can complicate an iritis or an iritis can complicate an existing glaucoma. In general, iritis is an inflammatory disease while glaucoma is not.

It is proper to treat the iritis without neglecting the glaucoma. The patient should be confined to bed in a darkened room; the diet must be light and sparing; elimination by the bowels, kidneys and skin must be encouraged. Salicylates are given intravenously or by rectum to toleration, moist hot compresses are used frequently, and artificial leeches are applied to the temple on the affected side.

Iritis may be confused with scleritis or with episcleritis. The absence of the usual signs of iritis and the presence of a single lesion outside the cornea should attract attention from the uveal tract.

Treatment of iritis and of iridocyclitis is divided into causal and symptomatic.

The first indication is to dilate the pupil. Atropin is the best agent. It contracts the blood-vessels of the iris and ciliary body and relieves the congestion; it is soothing; it dilates the pupil and suspends accommodation. A drop of 1 or 2 per cent atropin sulphate solution is instilled every one or two hours until the pupil is dilated, and then frequently enough to maintain dilatation. The addition of 4 per cent cocain solution facilitates the action; this is omitted after dilatation is effected.

Should fresh solutions of atropin fail, a small particle of the alkaloid may be dropped into the conjunctival sac, and repeated if and as necessary. When the patient is seen daily this is a good method. One-fiftieth grain of the sul-

Differential Diagnosis of Iritis, Glaucoma, and Conjunctivitis

	Iritis	Glaucoma	Conjunctivitis
1. Lids	1. Edema of upper lid above lashes.	1. Edema or swelling of upper lid.	1. Lids stick from secretions.
2. Conjunctiva	2. Clear; may be injected over globe.	2. Chemosis; congestion.	2. Opaque; congestion on both lid and globe.
3. Secretions	3. Tears.	3. Tears.	3. Mucous or purulent; sticky.
4. Congestion	4. Zone of fine, straight pink or violet vessels bordering cornea and fading toward the fornix.	4. Ciliary and episcleral vessels; may be of conjunctival vessels.	4. Conjunctival; brick-red meshes movable over sclera, fading toward cornea.
5. Cornea	5. Sensitive; deposits on posterior surface.	5. Insensitive; steamy or cloudy surface.	5. Sensitive; clear after irrigation.
6. Anterior Chamber	6. Normal or deepened; aqueous cloudy; hypopyon.	6. Shallow; deep in iritis and buphthalmos.	6. Normal.
7. Iris	7. Swollen; exudate on surface obscures pattern; surface dull or muddy.	7. Slate color; periphery forward; may be congested.	7. Normal.
8. Pupil	8. Gray from exudate; reacts sluggishly; contracted: Older cases have irregular border and evidences of synechiæ.	8. Sluggish or fixed to light; dilated oval.	8. Normal.
9. Lens	9. Exudate and iris pigment on surface.	9. Gray-green reflex at times.	9. Normal.
10. Vitreous	10. Exudates from ciliary body; opacities or floaters.	10. Normal.	10. Normal.
11. Tension	11. Normal or diminished; may be temporarily high.	11. Markedly increased usually.	11. Normal.
12. Tenderness	12. Circumcorneal if ciliary body is attacked.	12. Over ciliary region.	12. Normal.
13. Pain	13. In globe, or radiating over branches of fifth nerve. Photophobia.	13. Severe over branches of fifth nerve; in globe; insomnia; constitutional symptoms.	13. Sensation of foreign body; sandy, burning, itching, smarting.
14. Vision	14. Impaired from exudate. Fields concentrically contracted.	14. Diminished; nasal fields narrowed; halos around artificial lights.	14. Normal or diminished from presence of secretion on cornea. (Irrigate.)
15. Optic Disk	15. Normal when visible.	15. Excavated at margin; vessels bend abruptly; halo around disk.	15. Normal.

phate in solution may be injected beneath the conjunctiva. Adrenalin chlorid solution 1 : 1,000 may be added to the atropin either by instillation or by subconjunctival injection; one or two drops are used at each treatment. Atropin is also used in an ointment.

Homatropin 2 per cent with cocain 4 per cent is too weak unless used very early. It is useful for diagnostic purposes; relief of pain and the demonstration of black spots on the lens offer presumptive evidence of iritis. In elderly people atropin should be used with caution, to avoid precipitating an attack of glaucoma; but these persons are not very liable to iritis.

Dionin in 3 to 10 per cent solution, alone or combined with atropin, may be instilled or injected subconjunctivally in one drop doses. It tends to relieve the pain. Hypodermic injection of morphin into the temple of the affected side usually relieves the pain temporarily. Leeching in the same location is said to be of value in relieving the pain and in enhancing the action of atropin. Moist hot compresses applied for fifteen minutes each two hours act favorably.

The hygienic management of the case will depend upon its severity. Dark glasses must be worn over both eyes, and all near work must be discontinued. The patient may have to be placed in a darkened room. Physical exertion may be harmful, and occasionally the patient requires rest in bed. The diet should be light and nutritious, with few or no condiments and no stimulating beverages. Alcoholic drinks are not permitted. The bowels must be kept active. Pilocarpin sweats are often employed as an aid to elimination in active stages. Resistance must be built up and maintained.

Constitutional or systemic treatment will be determined by the cause. Focal infections must be eliminated in the order of their clinical importance. A negative blood Wassermann must not be accepted without provocative preparation. Where there is clinical evidence of syphilis, the iritis is probably due to it. The therapeutic test for specific infection is not dependable, for many cases of clinically nonspecific iritis do well on antisyphilitic medication. The treatment consists of mercury, preferably by inunction, the intravenous injection of an approved arsenical preparation, and later potassium iodid.

Tuberculosis requires the usual methods for tests and treatments. The test should not be made by instilling tuberculin into the conjunctival sac in cases of iritis; the reaction is too violent.

Gonococcal infection may be disclosed by the complement fixation test. A history of gonorrhea and a discharge from the urethra or a preceding or coincident arthritis constitute presumptive evidence. The prostate gland and seminal vesicles should be examined, but massaged very lightly if at all; the iritis might be aggravated. Other infections may coexist. Gonococcus vaccines are indicated.

Tonsillar infections are demonstrated by visible evidence and by cultures of the contents or secretions. A tonsil cannot be excluded as a factor when there is undue redness or swelling of the anterior pillar. Where there is a history of tonsillectomy, the anterior pillar must be pulled aside and the fossa inspected for pus or unhealthy tonsil remnants.

The teeth need to be examined. Exposed stumps, buried roots and devitalized teeth should be roentgenographed. Pyorrheal or other secretions may be cultured. The accessory air cavities of the nose are difficult. Tenderness over any sinus attracts attention. Pus in the middle nasal meatus generally comes from some air cavity. Transillumination and roentgenographs are often negative when clinical signs and symptoms are present.

Practically all of the important sources of focal infections have been considered. The removal of such a focus is indicated as soon as it can be done with safety. An ill-timed operation might further damage an acutely inflamed eye. The extraction of an abscessed tooth, or a tonsillectomy has been followed by almost immediate relief from iritis in some instances, while in others either operation has been followed by marked violence in the inflammation.

Sodium salicylate is indicated in large doses in the focal infections, including those due to gonococci. This may be given by mouth, by rectum or intravenously. The stomach will hardly tolerate a sufficient amount, even when combined with sodium bicarbonate. The rectum will tolerate 3 to 6 grams (grains xlv-xc) twice daily; it should be injected only into the lower or middle rectal pouch to obtain the maximum benefit. The intravenous daily dose is from 2 to 4 grams (grains xxx-lx); it is most effective when given in this manner, not only on the iritis, but for the control of pain.

Sodium iodid, in the same dose, may be combined with the salicylate, or it may be given alone later on to promote the absorption of exudates. Sodium bicarbonate is indicated when the urine contains diacetic acid or acetone.

Subconjunctival injections of 0.3 to 1 cubic centimeter (5 to 15 minims) of normal salt solution are given after the subsidence of inflammatory symptoms to encourage the absorption of exudates. It may be given every second or third day. Instillations of 1 drop of 5 per cent dionin solution once or twice daily is helpful. Mercury, potassium iodid, sodium iodid intravenously, and pilocarpin sweats are also used for the same purpose.

Operative treatment is not often necessary. Paracentesis of the cornea is indicated to diminish tension and to evacuate the hypopyon. This operation is done under local anesthesia with a keratome or a cataract knife. The lids are separated with the speculum or the retractors. The conjunctiva is seized with fixation forceps near the upper border of the cornea, and the incision is made below near the limbus. By depressing the lower lip of the wound the

exudates may be evacuated. The wound should be opened daily as long as necessary.

Where tension is maintained above normal, an iridectomy is indicated. It should not be done while the inflammation is very active, unless other measures have failed. An iridectomy is indicated in occlusio and seclusio pupillæ, and wherever the aqueous cannot enter the anterior chamber. In iris bombé an iridectomy is impracticable, because the periphery of the iris lies against the cornea. Transfixion of cornea and iris is practiced for this. A narrow-bladed cataract knife is used.

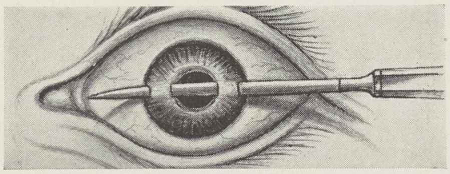


Fig. 137.—Fuchs' Operation for Iris Bombé. (After Meller.)

The lids are separated and the fixation forceps grasps the conjunctiva at the lower nasal border of the cornea. A puncture is made in the horizontal meridian of the cornea, well toward the limbus; it will also pass through the iris. The knife is carried across parallel to and in front of the natural plane of the iris to avoid injury to the lens. It will puncture the iris on each side of the pupil, and the counterpuncture will pass through the iris and cornea toward the nasal limbus (Figs. 137 and 138). The openings are permanent.

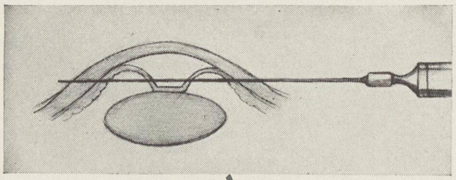


Fig. 138. (After Meller.)

Enucleation is indicated for a blind eye when the pain cannot be controlled, or where an intra-ocular tumor can be reasonably suspected of having caused the uveitis. Where an affected eye resists all efforts to reduce the inflammation and continues painful, and the fellow eye shows irritation, sympathetic ophthalmia must be considered most carefully.

TRAUMATIC IRIDOCYCLITIS AND SYMPATHETIC OPHTHALMITIS

The danger zone of the eye corresponds to a band 5 to 7 millimeters broad surrounding the cornea. The ciliary body reacts violently to injuries, and this reputation earned the name "danger zone." Simple contusions excite a hyperemia, but in the absence of infection the manifestation subsides under treatment by hot or cold compresses, atropin, and dressings.

Exogenous infections occur when organisms enter the eye through wounds of the conjunctiva and sclera. If the ciliary body is lacerated, an infection follows in almost every case. The organisms may accompany the injuring body

or object, or they can be driven in by it, or later migrate in from the conjunctival sac. This can happen following operations that open the eyeball.

The course and intensity of the reaction varies. Where the wound is light, healing soon occurs. In the most severe cases the onset of inflammation is rapid, the aqueous chambers quickly fill with exudate, pus forms and the eyeball is speedily destroyed. The earlier signs are those of severe iridocyclitis; the later are those of endophthalmitis. Coincidental injury to the lens often hastens the dissolution of the eye. In other cases the lens is absorbed and the eye remains quiet. After some weeks the globe softens.

The exudate consists of round cells and fibrin and is plastic in character; organization takes place in the vitreous chamber, and this leads to contraction or shrinking of the mass. The retina is detached and the eyeball softens. Where organization is particularly persistent, a dense fibrous tissue replaces everything inside the sclera, and the globe undergoes atrophy or phthisis bulbi. When these shrunken eyes are very painful they should be removed.

The treatment requires the removal of a retained foreign body when it can be done without added damage to the eye. Lacerated wounds require scleral or at least conjunctival sutures. The conjunctiva should be drawn over any perforating wound.

Hot or cold applications are used according to the degree of pain and inflammation present. The local treatment is the same as for iritis, with appropriate hygienic management. Rest, preferably in bed, light diet, and the avoidance of excitement are advised. Stimulating beverages must be prohibited.

Sympathetic Ophthalmitis, or sympathetic iridocyclitis or sympathetic ophthalmia occurs in the second or uninjured eye. It is practically always dependent upon a traumatic iridocyclitis in the first or injured eye. The occurrence of an iridocyclitis in the second eye is suggestive, but not conclusive of sympathetic inflammation. Its appearance there may be independent of that in the injured eye. A constitutional disease like syphilis may affect one eye and later the other; this is simply a bilateral manifestation of the primary disease.

The perforation of a corneal ulcer, or the changes that take place in the cells of an intra-ocular tumor may cause an iridocyclitis. Either of these conditions may bring about a sympathetic ophthalmitis. The first, perforation of a cornea that is ulcerated, may introduce an exogenous infection. Of the second class, the tumor is a choroidal sarcoma in most instances. It is thought that this neoplasm grows from the same kind of cells that are affected in sympathetic ophthalmitis; this is significant of pigmented cell sensitization.

The cause of the disease appearing in the second eye is not known. Every

theory thus far advanced has met an unexplained objection. For instance, panophthalmitis destroys the uvea, but does not cause inflammation in the second eye; not every traumatic iridocyclitis is followed by the disease in the second eye. The nature of the injuring substance seems to be a factor. Sterile glass rarely causes inflammation. Copper, even when aseptic, seems to irritate the uveal tissues and inflammation usually follows.

Sympathetic *irritation* of the second eye is not to be confused with sympathetic inflammation. Such irritation occurs in nearly every case of eye injury, and it is noticed to some extent in every case of iridocyclitis. It depends on reflexes from the trigeminus nerve, and may be called a neurosis. The symptoms of irritation in the second eye are lacrimation, photophobia, pain on attempting to read or do near work, neuralgic pain in the brow or orbit, and vascular congestion around the cornea.

The absence of symptoms of inflammation does not exclude the possibility of sympathetic ophthalmitis, and their advent must not be awaited before instituting treatment which is otherwise indicated. When the first eye is useless and the second is conspicuously irritated, the first must be removed.

In sympathetic *ophthalmia* the second eye may recover with little damage, or it may be lost regardless of treatment. Both eyes must be closely watched during the progress and subsidence of iridocyclitis in either one. The disease seldom attacks the second eye sooner than twenty days after injury to the first, but it has been reported in fourteen days; it has been known to occur after the lapse of forty years, so there is no period of immunity. The usual time of onset is from the fourth to the twelfth week, and this is the dangerous period.

The inflammatory manifestations in the first eye must be studied continually. The appearance of a plastic exudate portends danger. Tenderness over the ciliary body adds to the apprehension, but it may be absent. The nature of the wound may cause anxiety. When the iris, lens capsule or ciliary body is injured, more especially where one of them is caught in the wound, the danger is increased. The involvement of the second eye is independent of any loss of vision in the first.

The second eye is usually attacked insidiously. The earliest evidence may be iritis, fine deposits on the posterior surface of the cornea or optic neuritis. In these cases the disks are hyperemic and their margins are woolly; in the common form of optic neuritis the blurred zone extends a short distance beyond the disk. The retinal veins are engorged. Opacities form in the vitreous and obscure the fundus details, particularly the signs of neuritis. Vision is diminished. The pupil is contracted, but synechiæ do not form early or rapidly. The circumcorneal vessels are usually engorged. Headache is usually present and may be severe.

The disease pursues one of two courses. In one, the progress is slow and intermittent; the symptoms are mild and soon subside. After a time there is another attack, more severe than the first; each recurrence adds to the damage. Ultimately vision is materially affected or the eye may be lost.

In the other type the onset is rapid; plastic exudates quickly form, although hypopyon is uncommon, and when present is sterile. Annular synechia with iris bombé, or total posterior synechia often forms. The intra-ocular tension is elevated, and miotics are useless. Iridectomy is ineffectual, since the coloboma is soon clogged with the exudate, and the traumatism from the operation adds to the trouble. As the tension goes down the globe softens and ultimately atrophies. The whole process may occupy several months. When the disease is ushered in with severe pain, conjunctival chemosis and edema of the lids, an early atrophy of the eyeball may be expected.

A comparatively mild type of case is one in which there are some deposits on the posterior surface of the cornea, and some adhesion of the pupillary margin of the iris to the lens. The anterior chamber is deep. In another type, multiple small yellowish areas of exudate are found in the choroid.

Treatment of sympathetic ophthalmitis begins with prevention. Where the first eye is hopelessly injured it must be removed; when it becomes infected and plastic exudates begin to form, it should be removed. Later, when blindness seems to be inevitable, enucleation must be considered. When the globe is shrunken, painful or sensitive, or subject to recurrent inflammation, it should be enucleated. While removal offers best assurance against the development of the disease, immunity is not certain. The second eye may become involved after the first has been enucleated, although it rarely does so after four weeks have elapsed since operation.

As soon as the second eye becomes affected the first should be removed if it is blind to form, soft and diminished in size, or has a permanently opaque cornea. Since a highly inflamed second eye may go on to atrophy regardless of treatment, no first eye should be sacrificed if there is a reasonable prospect that it may be restored to usefulness.

The medical treatment of sympathetic iridocyclitis is empirical. Sodium salicylate is given in a daily dosage of 0.065 gram (grain i) of the drug to each 450 grams (pound) of body weight if the patient can tolerate it. It is usually combined with an equal amount of sodium bicarbonate. It has a more pronounced effect with less dosage when given intravenously. It can be given by low enema. Salvarsan has been given intravenously with excellent results. Mercury and potassium iodid are also used. The drug selected must be used boldly. Atropin is indicated.

When the inflammation in the second eye has been managed successfully,

the patient must be informed of his precarious condition and urged to report periodically for examination.

INJURIES AND THEIR TREATMENT

The iris may be injured by blows or contusions and by perforations. The former may dislocate the lens and the iris or lacerate the iris. Hemorrhage into the anterior chamber makes it impossible to examine the structures behind the cornea; atropin, a snugly fitted bandage and iced compresses are indicated.

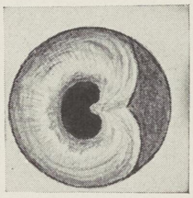


Fig. 139.—Iridodialysis.

An iridodialysis is a partial separation of the iris from its peripheral attachment (Fig. 139). Through the opening, which looks black to inspection and to oblique illumination, the red fundus reflex may be seen with the ophthalmoscope. When the ciliary body prolapses between the cornea and the detached iris, or when a tumor of the ciliary body detaches the iris there can be no fundus reflex (Fig. 140).

The iris is attached at the periphery only; if anything gives way it must be at this part. The pupil is flattened on the side nearest the detachment; it is D-shaped. Atropin is recommended to paralyze the sphincter and allow the detached border to approach its natural position; it seldom succeeds. Iridotasis is advised; the cornea is incised in the plane of the iris, the detached portion of iris is drawn into the wound to become incorporated with the corneal cicatrix; sutures may be used if desired.

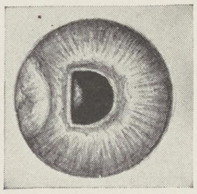


Fig. 140.—Tumor of Ciliary Body.

An absence of the iris is known as irideremia or aniridia. This cannot be repaired. Incomplete detachment is sometimes found congenitally (Fig. 141).

A blow or contusion may paralyze the iris sphincter; the pupil dilates. If the pupillary border of the iris is torn the pupil tends to dilate and separate the margins of the wound. Atropin is contra-indicated in the absence of irritability. Eserin in 0.25 per cent solution is instilled to contract the pupil.

Should an injury perforate the cornea, the aqueous escapes and the iris floats into the wound; it is rarely possible to completely replace the prolapsed tissue. When the prolapse occurs near the periphery of the cornea and is very large, the ciliary body may be included; it should be reduced if possible. Eserin may succeed in pulling part of the iris tissue away from the wound; the remainder should be cut off. The wound is closed with a conjunctival flap. Continued use of eserin may prevent the formation of an adherent leukoma.

Should the prolapse be more nearly central in the cornea, a mydriatic is

indicated to pull the iris away from the wound. Otherwise the treatment is the same. It is often advantageous to separate the conjunctiva from the limbus, undermine it for a distance outward, and bring it over the wound with a purse-string suture. The suture is removed after four or five days. The mydriatic is continued while the flap is in place.

Small penetrating bodies may injure the cornea without loss of aqueous. These may perforate the iris or become imbedded in it. Where imbedded they can be removed with forceps after making a small incision near the periphery of the cornea. Magnetizable bodies should be dislodged before the corneal incision is made; they are then withdrawn through the incision with the magnet.

A perforation of the iris does not heal. An opening found in a case of recent injury may have no corresponding fresh wound in the cornea; an inquiry should be made about former accidents. Corneal scars should be searched for in new injuries. These relations have industrial compensation importance. Atropin, White's ointment and eye pad are indicated.

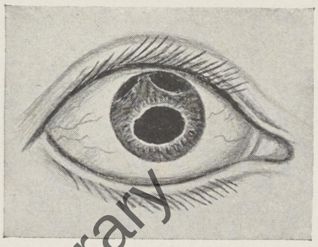


Fig. 141.

Siderosis follows the chemical changes that occur in a retained particle of iron. It consists of a greenish or rusty brown discoloration of the iris.

The ciliary body can be injured by contusion or laceration. Hemorrhages occur into the aqueous or vitreous chambers. Cyclodialysis is a detachment of the ciliary body from the sclera; it opens a way for aqueous to enter between the choroid and sclera and possibly detach the choroid. Surgical cyclodialysis is performed in the treatment of glaucoma. In these separations the detached part approaches the lens and relaxes the fibers of the zonule. This has the same effect as a spasm of accommodation, and is the opposite of a paralysis; consequently, myopia may be a symptom.

A wound passing through the "danger zone" involves the ciliary body. A subconjunctival or intra-ocular hemorrhage occurs and the ciliary body may prolapse into the wound. Separate sutures are required for the sclera and the conjunctiva. Intact conjunctiva must cover the scleral wound. Atropin, antiseptics, bandage, iced compresses and rest are indicated. Severe injuries may require immediate enucleation.

ANOMALIES OF THE IRIS

Melanomata are identified as spots of brownish color scattered irregularly over the surface of the iris; the condition is usually bilateral.

Persistent Pupillary Membranes are generally found as brown hairlike threads extending from one part of the iris to another in the region of the pupil. The threads are attached outside the sphincter zone, or to the pupillary ends of the radial fibers of the iris (Fig. 142). They may stretch across the pupil, or form a chord or network on one side of the pupil. They are unabsorbed remnants of the fetal vascular membrane that covers the surface of the iris and lens.

Additional evidence of incomplete absorption is found as congenital pigment on the lens. This pigment appears as very fine dots arranged more or less regularly within a circular *area* corresponding to the pupil. These can be distinguished from inflammatory processes which leave a *ring* or a part of a ring of dots on the lens; this ring corresponds to the pupillary border.

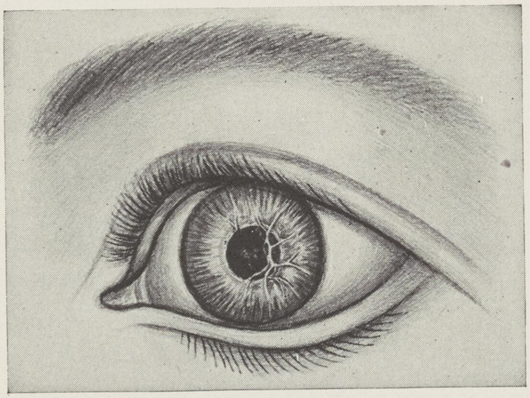


Fig. 142.—Persistent Pupillary Membrane. (After Parsons.)

Corectopia means a displaced pupil. The normal pupil is usually situated slightly downward and nasally from the center of the iris. In corectopia the displacement is greater, and the pupil is unlikely to be circular in outline.

Polycoria is a condition of multiple pupils (see Fig. 141); multiple images may form on the retina, one from each opening.

Coloboma of the iris is an extension of the pupillary aperture in the direction of or quite to the periphery of the iris. It is usually directed downward, and is generally bilateral. Its counterpart is an artificial coloboma formed by iridectomy. In the congenital form the sphincter zone is preserved. Colobomata of the iris, ciliary body and choroid are often associated.

Rarely the pupils are found to be slitlike, similar to those of the cat. At times the pigment on the border of the pupil rolls out as an ectropion. Where this border is nodular, each body may resemble a corpus nigrum bordering the pupil of the horse. Cysts of the iris occur.

A regular lacelike pattern of fine white lines is often seen on a light iris; it may be due to sclerosis of superficial vessels.

THE CHOROID

Essentials of the Anatomy.—The choroid is the third part of the uveal tract. It is rich in pigment and forms a dark lining for the globe. It extends

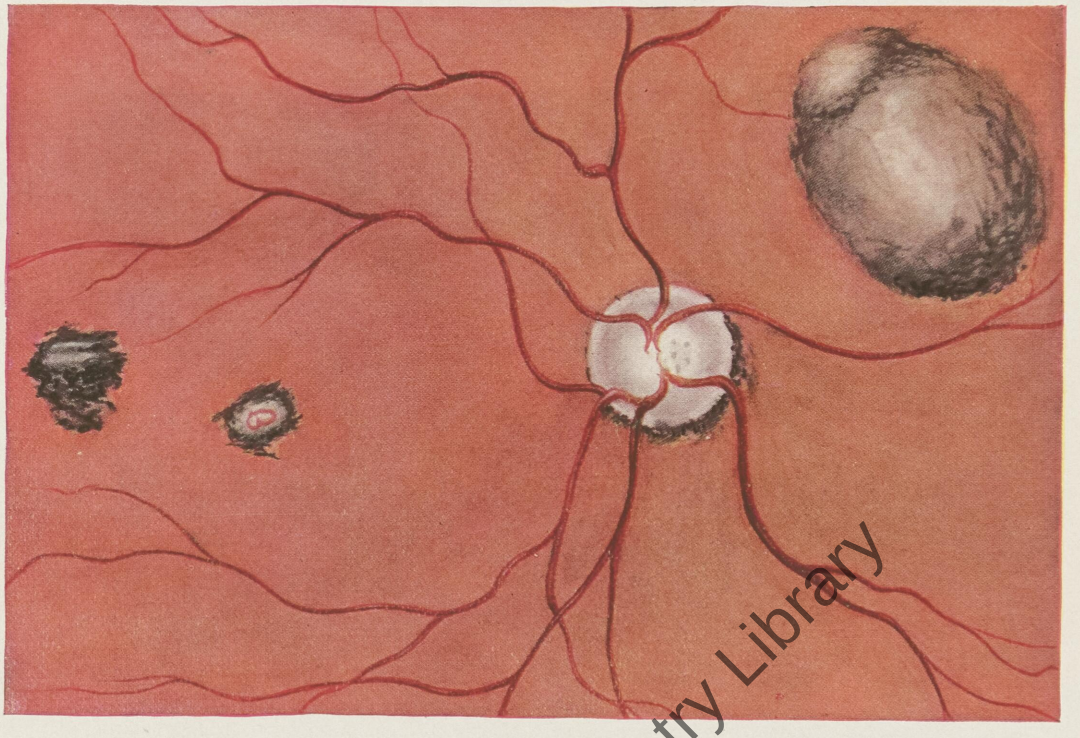


Plate VII.—Tuberculous Chorioretinitis with Tubercle of Choroid, Macular Degeneration and Atrophy of Disk.

Right eye, advanced. Female, aged fifteen; Wassermann tests negative, tuberculin tests positive; healed lesions in lungs; chronic tuberculosis. (Dispensary patient.)

from the margin of the scleral foramen or the optic disk forward to its junction with the ciliary body. The choroid is composed of five layers; the outer or suprachoroid is a fibrous tissue which blends loosely with the lamina fusca of the sclera; the next three layers are vascular; the inner layer is a homogeneous membrane called the lamina vitrea, which is somewhat similar in appearance to Bowman's and Descemet's membranes in the cornea. The last is in intimate relation with the pigment cells of the retina.

Of the vascular layers (Fig. 143) the outer is the layer of large vessels. These are veins which converge into four or more trunks, the venæ vorticosæ, which pass obliquely backward and outward through the sclera near the equator of the globe. These trunks form the superior and inferior ophthalmic veins. The middle layer is composed of medium-sized vessels. The inner layer is the choriocapillaris, or layer of capillaries. The red fundus reflex is due to the blood in the choroid.

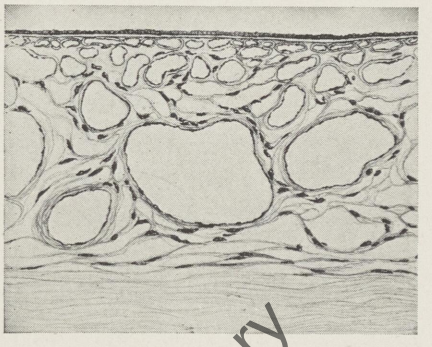


Fig. 143.—Vessels of the Choroid. (After Fuchs.)

The vessels seldom anastomose in the different layers. They are supported by connective tissue. The intervascular spaces are well occupied by pigmented cells in the layer of large vessels, but sparingly in the middle layer; the vessels are so numerous in the capillary layer that the spaces are negligible. The arterial supply for the choroid is from the short posterior ciliary arteries. These enter the scleral coat in an irregular circle about the optic nerve. The nerve supply is from the ciliary ganglion.

The choroid is attached to the sclera at the scleral foramen and it unites with the ciliary body. It is also attached where its arteries enter it from the sclera, and where the venæ vorticosæ pass through the sclera from it. Otherwise there is a potential space between the choroid and sclera. Hemorrhages and other fluids can separate them.

The choroid is subject to the same diseases that affect the iris and ciliary body. It is often involved with them in one pathological process. When the choroid is known to be affected, involvement of the iris and ciliary body is suspected when there are changes in the iris, engorgement of deep circumcorneal vessels, precipitates on the posterior surface of the cornea, reddened eyeball, swelling of the lids, tenderness over the ciliary body and symptoms of irritation. This is called uveitis or iridochoroiditis.

The choroid is coextensive with the retina. They extend from the optic disk to the ora serrata. The choroid supplies blood to the outer layers of

the retina. The pigment layer of the retina clings to the choroid when the retina is detached.

The rod and cone layer of the retina lies upon the pigment layer. These delicate rods and cones are the essential cells of visual perception. An affection of the choroid produces a change in a corresponding area of the overlying retina. A chorioretinitis denotes a primary change in the choroid and a secondary change in the retina. A primary affection of the retina may cause a secondary change in the choroid, and this is called a retinochoroiditis.

Choroiditis occurs in two forms, nonsuppurative or exudative, and suppurative or purulent. The former pursues a chronic course which is mostly limited to the choroid and the retina, while the latter is an acute process which soon affects the ciliary body and iris and may involve the remaining contents of the globe. The suppurative form is regarded as an endophthalmitis or a panophthalmitis. An endophthalmitis is a purulent exudate in the vitreous; it is a panophthalmitis when it involves the remaining structures, breaks through the coats of the globe and discharges externally.

Choroiditis when due to special causes is named according to an etiological classification, as syphilitic, metastatic or myopic. It is also defined according to location as anterior or central, and according to distribution as disseminated or diffuse.

Exudative Choroiditis appears first in single or multiple foci of inflammation. These foci are seen as small creamy spots in contrast with their surroundings, or as fairly large buff-colored areas that merge into the surrounding tissues with indistinct outlines (see Plate VIII, Fig. D). In the former case the media of the eye are usually clear, while in the latter there appears to be a mist or a clouding of the media which is intensified over the various affected areas. The effect is to conceal or disguise areas of the eye-ground.

The vessels of the retina are seen to lie in front of the patches of exudate. These vessels seem to bend slightly as they pass over the exudates, for in such places the retina is edematous, and because of the infiltrative process the choroid is thickened. The elevation is not usually pronounced. The surrounding retina and choroid appear healthy.

The exudates are resolved by absorption. The pigment disappears from the central part of the lesion and leaves a pale area which gradually becomes white. When such a spot reflects a tint of blue the sclera is exposed. Without such suggestion of blue the spot is white from connective tissue proliferation. Traces of choroidal vessels may be seen in it.

Usually the uveal pigment also proliferates and piles up as a jet-black ragged border around the area; it may invade the retina and cover parts of its vessels. The borders of pigment are irregular or roughly circular in outline.

The fundus sometimes looks like a map. The sizes of the patches vary; when small they are often entirely covered with pigment, while spots of pigment often appear on the floors of the larger areas. The retinal vessels pass over the cicatrices without change of width, and with little or no change of level.

The appearance and color of the patches afford a clew to their age. While cream or buff they are recent. When they become pale they are from several months to a year old. When they show white or bluish white centers sharply bordered with black they are not less than two years old; this stage undergoes little further modification. In many cases the various stages will be found coexistent. The patches often coalesce.

In some rare cases the disease is evenly diffuse, and very mild in its destructive tendencies. The pigment of the retina may disappear and leave the choroid almost intact. In such instances the larger choroidal vessels are visible, and the appearance is similar to a type of eye-ground known as the tessellated fundus. A former disease such as syphilis can be inferred when this pattern is unusually clear. The choroidal vessels are plainly visible in retinitis pigmentosa, and occasionally in cases of glaucoma and myopia. The choroid has been more profoundly diseased when some of its vessels show sclerosis of their walls or when they have been replaced by white cordlike connective tissue. Other evidences of sclerosis are often present.

Changes occur in the vitreous, and opacities or "floaters" are produced. These may or may not interfere with vision. They may resemble muscæ volitantes subjectively. Muscæ cannot be demonstrated objectively, but genuine opacities can be. They may be seen as fine threads, crumpled hair, or small specks to large black masses. They are free and change their positions with movements of the head and eyes. To demonstrate the floating opacities the patient is taken into a darkened room and directed to look steadily at some definite object. It is sometimes necessary to dilate the pupil.

With a plus 7 lens in the sight hole of the ophthalmoscope, the light is directed through the pupil toward the optic disk, which is usually the lightest part of the fundus. The point of observation is from 12 to 15 centimeters (5 to 7 inches) from the eye, about 15 degrees to the temporal side of and in the same horizontal plane with it. By varying the distance and position between the observed and observing eyes the pale background of the disk can be located. The patient is instructed to look alternately and rapidly at the ceiling and floor, and again steadily at the object in front of him.

When the eye is brought to rest the floaters will be seen to slowly settle toward the lowest part of the fundus. The finer the opacities the more slowly they sink, and the coarser they are the more rapidly. The rate of sinking does not depend entirely upon the size and density of the floating bodies. The

vitreous is affected by the process that produces the exudates. In general, the fluidity of the vitreous is in accord with the mass of the floating body; a large, rapidly sinking body suggests a very fluid vitreous. The moving bodies may be in any part of the vitreous chamber. By repeating the exercise with different lenses, each zone of the vitreous may be examined.

Floating bodies often appear in a fine form from long-continued focal infections. They are common in cases of high myopia. They are present in undetected degenerative changes in the choroid. They generally appear black, but when very fine they may appear brown or gray. When they are white like snowballs it is asteroid hyalitis; when they sparkle like tinsel it is synchysis scintillans. In the last two conditions the vitreous is quite fluid. It is probable that the choroid is diseased to some extent when floating bodies of any kind are present. When floating opacities are projected into the field of vision, the patient can learn to rotate the eye and shift the opacity.

Choroiditis Juxtapapillaris or Jansen's disease is found next to or in the vicinity of the disk. The pigment is not sharply heaped up nor so black. It has been observed in cases having a remote focus of a low-grade infection. Corresponding scotomata appear in the visual fields.

Tuberculous Chorioretinitis (see Plate VII) is of an inflammatory character. The lesions are variable in size and affect the posterior portion of the fundus, including the macular area. The lesions are progressive, probably from a tubercle in the choroid. Large areas of choroidal edema are finally absorbed and leave a dull white layer of tissue on the sclera. In these cases there is usually a history of tuberculosis, possibly x-ray evidence of healed lesions in the lungs, negative Wassermann and positive tuberculin reactions. The treatment is by tuberculin.

This condition must be differentiated from chorioretinitis due to other causes, principally syphilis. In the latter the lesions are harsh and sharp; in the former they are soft and smoky until the stage of resolution. After that stage it may be difficult or impossible to differentiate the types ophthalmoscopically.

When these lesions are found in one eye only they are probably congenital, and are due either to developmental defects or to intra-uterine disease.

Central Choroiditis is limited to the macular region (Plate VIII); the usual changes are demonstrable. In early stages the macula often has a metallic appearance. It is usually bilateral, but not necessarily to an equal degree. It may appear in one eye while an extramacular lesion appears in the other. Central vision is diminished or lost.

Myopic Central Choroiditis is due to a stretching of the sclera and choroid at the posterior pole of the eyeball. Degeneration is manifested as a

choroiditis. The choroidal pigment does not degenerate, but rather tends to proliferate; while the area may become pale, it is more inclined to be distinctly dark. When the degeneration assumes an atrophic character the area may look like an ordinary choroidal lesion with a white center. Small hemorrhages often occur in or near the macula. Both eyes are usually affected. Central vision is diminished.

In central choroiditis due to infection or to myopia, the lesion is not usually larger than the disk. A blow on the eye may produce central changes with some resemblance to the myopic lesion; it is unilateral.

Coloboma of the Macula is usually unilateral and a little larger than the disk; it may resemble a central choroiditis. Where myopia and infection are absent, a lesion is probably due to a congenital defect. Other defects should be looked for.

Central choroiditis might be assumed in cases where the macular region is studded with yellowish dots. This is apparently a colloidal change that is not well understood. In one patient both eyes appeared to be symmetrically and equally affected; one eye was blind and the other had good vision. Guttate choroiditis attacks elderly people. The dots about the macula are white or creamy; they represent colloid excrescences formed on the lamina vitrea, and do not diminish vision.

Peripheral Choroiditis is characterized by lesions situated so far forward that they cannot be seen clearly without dilating the pupil. The pigment is prominent, but there are few white spots.

Symptoms of choroiditis consist of defects of vision and subjective sensations of light. Vision is affected according to the location of the lesions. It is diminished because of edema of the retina and the vitreous mist. The light and color senses are reduced. Night vision is frequently diminished. Images are often distorted.

The infiltration in the choroid elevates the retina over each affected area. This elevation alters the normal relations of the distances between neighboring rods and cones. Where these elements are separated the image of an object is diminished in size, and where they are crowded together the image of an object appears magnified. Images are distorted when the surface of the elevation is irregular; some elements are crowded while others are separated. When traction is made on the retina, as by some change in the choroid, the visual elements are mechanically stimulated; this causes subjective sensations of flashes or sparks of light.

The course of the disease is toward blindness. The presence of old and new foci in the same fundus demonstrates that the disease is both continuous and progressive. Opacities in the vitreous indicate the permanent character

of the changes. Impaired nutrition or toxins lead to atrophy of the retina and optic nerve and to clouding of the lens.

Exudative choroiditis is caused by congenital and acquired syphilis and by focal infections; it is sometimes found in chronic diseases that are marked by lowered nutrition. The cause in some instances cannot be determined.

Treatment of exudative choroiditis is both symptomatic and according to the cause in each instance. The patient should wear glasses to correct ametropia. Dark lenses protect from bright light. No near work should be done. Potassium iodid, mercury, arsenicals intravenously, subconjunctival injections of normal salt solution and instillations of dionin are used to promote absorption of the exudate. Where tuberculous infection is the probable cause, tuberculin is indicated.

Recent cases have a good chance for arrest or improvement, and some of them recover. Advanced cases offer little more than the prospect of arresting the progress of the disease; degenerated and atrophic tissues cannot be restored. Atrophic processes in the retina and optic nerve can sometimes be arrested by strychnin and nitroglycerin; the strychnin must be given in ascending doses up to toleration, and the treatment must be continued over a long period of time. These drugs seem to have little effect on the diseased choroid.

Suppurative Choroiditis is also known as *purulent choroiditis* and *metastatic choroiditis;* where the iris and ciliary body are involved it is called *suppurative or septic uveitis* or *iridochoroiditis.* It affects one or both eyes. It is caused by pyogenic organisms; these are brought to the choroid by bacterial or septic emboli or by metastasis, or they gain entrance in penetrating injuries.

Endogenous infections come from any disease in which pyogenic organisms are carried in the blood stream. The acute exanthemata, ulcerative endocarditis, pyemia, puerperal fever, and cerebrospinal meningitis may cause this disease. Exogenous infections accompany or follow accidental or operative opening of the eyeball. The retention of a foreign body in the eye is frequently followed by suppurative choroiditis. A foreign body infection often begins as an abscess of the vitreous, although a vitreous abscess may exist without profoundly involving the choroid.

Except in those cases in which the vitreous is primarily infected, the exudates are formed in the choroid first. From the choroid they invade the vitreous. When the disease is confined to the structures behind the lens it is called *endophthalmitis.* It is called *septic* uveitis when it has invaded the ciliary body and iris. Septic uveitis leads to shrinking of the globe.

In very light cases the choroid is only mildly affected, the retina is edematous and the vitreous is hazy. There may be some vision for hand movements. The prognosis is fair.

Endophthalmitis.—The typical form of endophthalmitis occurs in some cases of cerebrospinal meningitis. Severe attacks of pneumonia may cause it, or it may appear in one eye from a focal infection. Nothing can be seen behind the lens except a grayish or yellowish substance, which may or may not have minute blood-vessels on its surface. This is particularly called "metastatic choroiditis"; it is nearly always symmetrical and bilateral. It has been confused with glioma of the retina, and is included in a group of intra-ocular diseases which are denominated "pseudoglioma."

The age of the patient and the history of recent meningitis are suggestive for the diagnosis. Glioma rarely occurs after the age of seven years, and nearly all cases occur before three. Both eyes are seldom involved at once.

The symptoms vary in intensity. They may be absent, except for impaired vision. The intra-ocular pressure is usually low. When the tension is high the pain will radiate over the head in an area corresponding to the distribution of the ophthalmic division of the trigeminus.

Treatment is designed to protect the eye and to carry off the exudate. Dark glasses must be worn and the pupil must be kept dilated with atropin. Autogenous or stock vaccines are indicated. Nonspecific proteins have not been used enough to qualify them definitely in the therapeutics of this disease. Severe cases must be kept in bed. Moist hot compresses are usually indicated, although iced ones may be more acceptable to the patient. Foci of infection are to be removed with due regard for the possibilities of anaphylactic reactions.

Surgical treatment is not ordinarily given as long as there is perception and projection of light. If vision is lost the disease is progressing badly, and the eyeball should be enucleated or eviscerated.

Panophthalmitis is a destructive septic inflammation of all of the structures of the eyeball. It leads to rupture of the globe and the discharge of its contents. It originates from the same causes that produce suppurative choroiditis and endophthalmitis. It is occasionally secondary to orbital cellulitis and perforating corneal ulcers.

The signs of the disease are edema and congestion of the lids and conjunctiva, redness of the eyeball, proptosis, and limitation of movement. The proptosis is due to edema of the retrobulbar tissues. The fixation of the globe depends upon a plastic exudate which unites the fascia bulbi to the sclera. When enucleating such an eye these structures are separated with difficulty.

In the earlier stages a gray or yellowish reflex is observed behind the lens; at this time it might be classed with the pseudogliomata. The intra-ocular tension is raised during the most active part of the disease. Rupture of the eyeball and the discharge of its contents are followed by shrinking of the globe or phthisis bulbi. Eyes shrunken from this cause are not usually tender.

Rupture of the globe occurs through the sclera near the limbus ordinarily, but sometimes through the cornea. Because of congestion in the anterior uvea and stasis in the circulation, the nutrition of the cornea is impaired. A common sign is a ring ulceration near the periphery, and a slough forms in a very few days; the center of the cornea remains fairly translucent.

Symptoms of the disease are loss of vision, intense pain, and the constitutional reactions to sepsis.

Treatment other than surgical seldom arrests the disease, although it may mitigate its severity. Irrigations of bichlorid of mercury 1 : 2,000, and the constant employment of compresses kept wet with this solution have seemed to be helpful in some cases. Paracentesis often does good; it relieves the pain temporarily. The surgical wound must be sprung open daily.

The pain demands attention; morphin may be injected into the temple of the same side, and it is well to combine it occasionally with 2 per cent novocain solution to relax the lids. An external canthotomy will relieve pressure on the globe. Due regard must be had for the surgical hazard of dividing edematous tissue in the vicinity of a septic process. Other pain-relieving measures are employed as indicated.

When the usual measures for controlling pain are ineffectual, the abscess must be removed. This is accomplished by enucleating the globe or by eviscerating its contents. These procedures are not recommended during the active inflammatory stage if avoidable.

The argument against enucleation is that pyogenic organisms can migrate intracranially by way of the optic nerve sheaths and produce meningitis, but it is doubtful if they may not be carried by metastasis. It is seldom possible to enucleate such an eye without rupturing its walls and contaminating the wound. The adhesions of the inflamed fascia bulbi do not permit safe or gentle manipulation. As a rule the symptoms of panophthalmitis subside rapidly after evisceration.

Coloboma of the choroid is typically situated at the site of the fetal ocular cleft, but can exist elsewhere. It may extend to the sheath of the optic nerve, and unite with a coloboma of that structure. It need not extend so far. Sometimes the coloboma is irregular, and extends toward the optic nerve by a chain of island defects. It is a triangular white area whose apex points toward the disk; it may or may not be crossed by retinal vessels, and brownish retinal pigment is often present.

A coloboma of the retina sometimes coexists; examination of the visual fields decides the presence or absence of it. Choroidal colobomata are unilateral or bilateral. They are often associated with other congenital defects. In one bilateral case in the Riley Hospital for Crippled Children the vision of

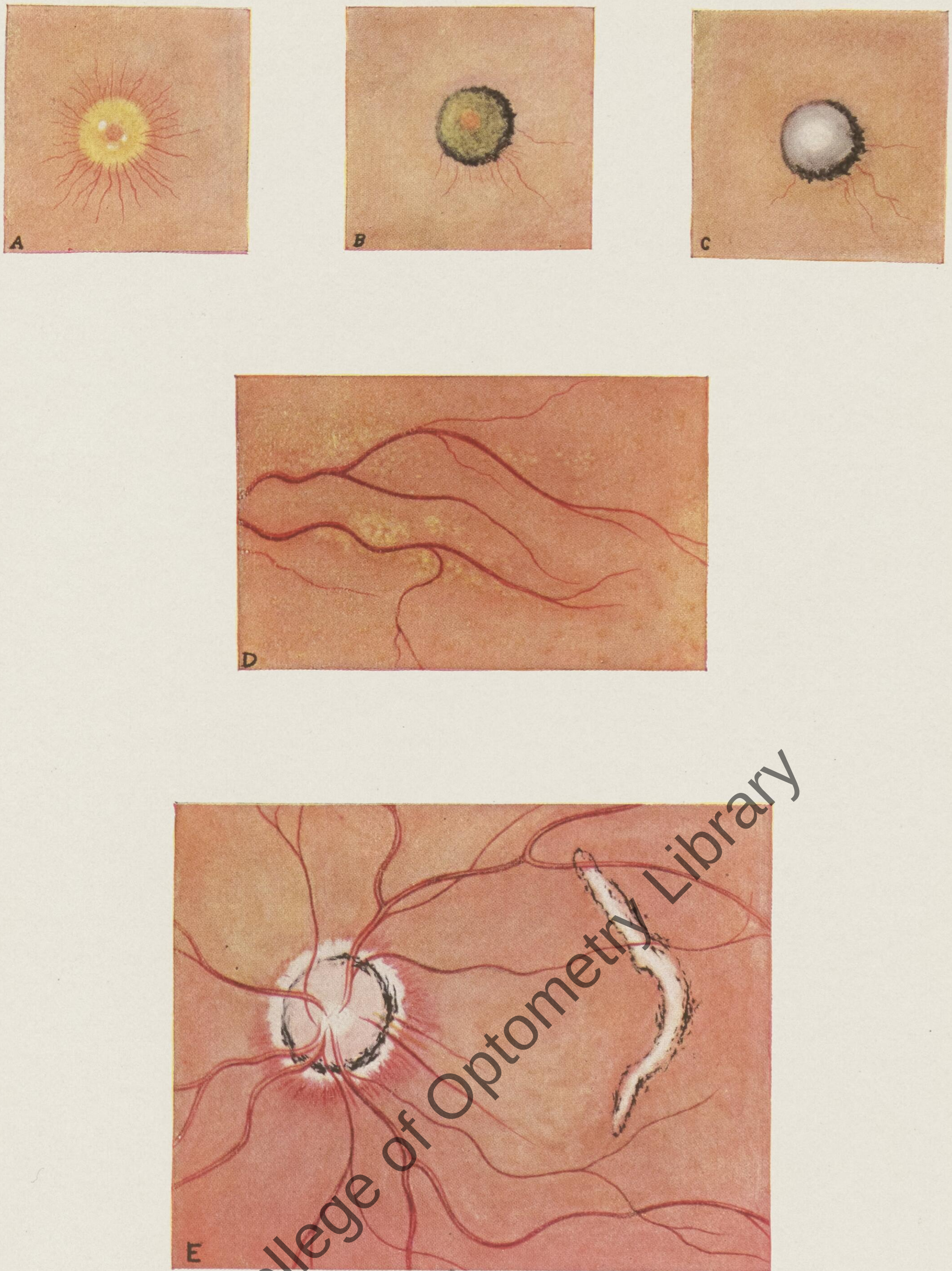


PLATE VIII.—CHOROIDITIS. RUPTURE OF CHOROID.

A, central choroiditis or chorioretinitis, right eye; *B*, central choroiditis or chorioretinitis, left eye; *C*, left eye, later stage, degeneration; *D*, chorioretinitis; *E*, rupture of choroid and partial evulsion of sheath of optic nerve, right eye. (Dispensary patient.)

the right eye was practically normal, while the left eye was microphthalmic and had 18 diopters of myopia with internal squint. The treatment is to secure best vision with appropriate glasses.

Rupture of the choroid is caused by injuries, usually contusions. Hemorrhage conceals the condition at first. Later, resolution occurs and the diagnostic figures can be made out. These consist of crescentic white lines or stripes roughly concentric with the border of the disk, and usually to its nasal side (see Plate VIII, Fig. E). Cicatricial bands often form and this is known as proliferating retinitis. Hemorrhage in an eye requires rest, preferably in bed. There is no other treatment for rupture of the choroid.

Detachment of the choroid follows a loss of vitreous, or a hemorrhage between the sclera and choroid. A hemorrhage is commonplace with detachment. The loss of vitreous may be due to trauma, or it may be lost during or following operations for glaucoma or for the removal of cataract.

Tubercle of the choroid usually exists in the miliary form. It resembles early disseminated choroiditis in that the spots are discrete and cover most of the fundus, but the lesions are only 1 or 2 millimeters in diameter by direct ophthalmoscopy. A single tubercle may be found in tuberculous chorioretinitis (Plate VII).

Tumors of the choroid are mostly sarcomata, and these are usually melanotic or characterized by pigment proliferation. Other forms of tumors have been identified (see Fig. 145).

Ossification of the choroid has been found in some eyes that became blind from inflammation.

Atrophy of the choroid sometimes follows inflammation or degenerations.

Albinism depends upon a deficiency of pigment in the iris and choroid.

CHAPTER X

THE RETINA

ESSENTIALS OF THE ANATOMY

The retina is the peripheral end-organ of light perception and vision. It is a thin, delicate transparent membrane coextensive with the choroid. Its pigment-cells and neuro-epithelium are nourished by the choroidal vessels. The pigment layer adheres to the choroid when the two structures are separated.

This pigment layer and a layer of retinal epithelium are continued forward, in a modified form, over the inner surfaces of the ciliary body and iris to the border of the pupil; they are not concerned with light perception or vision. The ganglion-cells, whose processes enter the optic nerve, are not found anterior to the ora serrata. The latter forms a wavy line and marks the anterior boundary of the retina.

The retina is composed of eleven layers. From without inward these are: (1) Hexagonal pigment-cells, (2) neuro-epithelium or rods and cones, (3) external limiting membrane, (4) external nuclear layer, (5) external reticular layer, (6) Henle's fiber layer, (7) internal nuclear layer, (8) internal reticular layer, (9) ganglion-cell layer, (10) nerve-fiber layer, and (11) internal limiting membrane. The retinal blood-vessels nourish all layers except the first five. The layers are bound together by the supporting neuroglial fibers of Müller; the external and internal limiting membranes appear to be formed by interlacings of fibrils from the outer and inner ends of Müller's fibers.

The retina is loosely attached to the choroid except at the ora serrata and macula, although the latter is a weak attachment. The fibers composing the nerve-fiber layer pass through the scleral foramen into the optic nerve, so the retina is held closely applied at that point also. In detachment of the retina the membrane retains its holds at the ora serrata and the optic disk, and occasionally at the macula. The retina is held in contact with the choroid by the pressure of the vitreous body.

The retina is about 0.042 millimeters thick at the border of the disk where the nerve-fibers are most numerous. The thickness gradually diminishes toward the ora serrata, where it is about 0.014 millimeters.

The fovea centralis (Plate X, Fig. A) is a circular depression, 0.2 to 0.4 millimeter in diameter, situated about 3 millimeters (15°) temporally from

the optic disk. It is surrounded by the macula lutea or yellow spot. Here the layers of the retina are reduced to pigment-cells, cones, few nuclear cells, many ganglion-cells and nerve-fibers, and the supporting fibers of Müller; the latter are laid very obliquely to the surface. At this point the retina is about 0.010 millimeter thick. The fovea is the area of sharpest vision, and is supplied entirely by the vessels of the choroid.

Cones only are found at the fovea. Proceeding from this locality, rods are more numerous and cones are correspondingly less numerous, until practically rods only are found at the ora serrata. Luciana (*Human Physiology,* Vol. IV, 1917, p. 335) states that there are about one million nerve-fibers, three million cones, and about six or seven times as many rods. Besides visual cells, the presence of light sensitive elements is proven by the contraction of the pupil in a blind eye that is exposed to light.

Vascular Changes.—The central artery of the retina is a branch of the ophthalmic. The central vein is made up of retinal branches and empties into the superior ophthalmic vein; the blood is discharged into the cavernous sinus of the same side. These vessels are classed histologically as arterioles. When viewed with the ophthalmoscope the blood column is seen, but the vessel walls are not visible in health. When the walls are thickened by sclerosis they are sometimes demonstrable.

The width relations of the arteries to the veins are as 2 : 3 or 3 : 4. The arteries are lighter in color than the veins, and their longitudinal centers are marked by a "light streak" which appears to divide the blood into parallel columns. The veins may be so marked also, especially near the disk, but not uniformly in all eyes. The veins usually cross under or behind the arteries. The vessels are accompanied by a plexus of sympathetic nerves. It is not certain that they are accompanied by lymph passages.

The optic disk is about 1.5 millimeters in diameter. At some point within its boundaries there is a depression which is called the physiological cup or the porus opticus. This cup has no fixed shape, outline or depth, and it does not normally extend to the margin of the disk at any point. The central artery enters the eye, and the central vein leaves it by way of this cup. Usually these vessels divide behind or in the cup, or at the level of the disk surface into superior and inferior branches. These subdivide into temporal and nasal branches, and by further arborization supply the retina.

The retinal arteries are terminal. The occlusion or attenuation of the central artery will affect the entire retina; where only one branch is impervious, a corresponding area of retina is affected. Occasionally a branch of a posterior ciliary artery enters the eye near the temporal border of the disk and extends toward the macula (Fig. 144); it is called a cilioretinal artery.

It will supply this limited area of retina and preserve its function when failure of the direct retinal circulation would result in temporary or permanent complete blindness. Unless this area can be kept alive and maintain communication between the macula and the disk, the choroidal nutrition of the macula will not save vision.

A branch of the central artery, given off behind the cup, may appear upon the surface between the cup and the margin of the disk. It resembles a cilioretinal vessel. Rarely a vein disappears at the margin of the disk and apparently enters the sheath of the nerve; it is called an opticociliary vein. It probably joins the central vein within the nerve sheath.

The central vein normally pulsates. This is most easily detected where the vein bends over the edge of the physiological cup to enter the nerve. Pulsation may also be seen where a large vein makes a sharp bend.

The central artery does not pulsate normally. When pulsation is present, it is detected where the artery bends out of the cup. Where intra-ocular pressure is lower than systolic and much higher than diastolic blood-pressure, the artery will pulsate. The phenomenon would be expected in aortic regurgitation, especially where the diastolic pressure is almost or quite nil; it does not appear in mild cases. Some mitral defects produce it. It is often seen in cases of anemia, exophthalmic goiter, and aneurysms of the aorta or carotids. This pulsation may or may not be present in glaucoma.

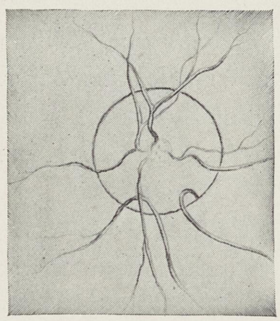


Fig. 144.

Pulsation should be looked for by the direct method of ophthalmoscopy. The patient's head and the hand that holds the instrument must be steady. While viewing the disk with the ophthalmoscope, pulsation may be produced by light pressure with the finger tip on the eyeball. Pulsation is rarely conspicuous and it must be looked for intentionally. When it is seen or suspected the rhythm is observed; gentle finger pressure applied very gradually will not change the rhythm. Too much finger pressure may obliterate the vessels.

Pulsation may occur in the usual way or as a slight movement of the whole vessel. This can sometimes be seen at a bend of the vessel at some little distance from the disk. When the disk becomes hyperemic and anemic rhythmically, it is significant of aortic regurgitation; the Corrigan pulse will be present.

The retinal arteries are seldom dilated unless the veins are also. The arteries are frequently contracted independent of any change in the veins. Engorged vessels are noticeably tortuous, while contracted vessels are inclined to be straight. Tortuous vessels may be partly buried in the retina or vitreous,

so that the blood column appears to be very irregular in diameter and color. Such vessels seem to be sacculated.

In states of engorgement many fine vessels, previously invisible, appear in the retina; this may be the earliest evidence of accessory sinus disease. In states of contraction many fine vessels, previously visible, disappear from view. This is common to the intra-ocular manifestations of several diseases.

Arteries are apparently contracted in arteriosclerosis; the walls are thickened by endarteritis and the blood column is narrow. The thickness of the wall may be estimated in many cases by noting the disappearance of a section of a vein where it is crossed by an artery. The arterial blood column accounts for a part, and the arterial wall for the rest of the missing section. The vein has the appearance of being pinched by the artery; it bends abruptly to cross the artery at nearly a right angle, its blood column is wider before crossing than after, it may pulsate at this point, and the section behind the artery is invisible. Pinching near the disk loses significance.

Sometimes the blood column has no central light streak but looks like a "copper wire" artery. A later manifestation is a "silver wire" artery which looks like connective tissue. These designations are used in stages of sclerosis.

Thickening of the vessel walls with diminution of the blood column is due to syphilis, chronic nephritis or cardiovascular-renal disease, and in some cases to diabetes. The vessels are highly contracted and straight in retinitis pigmentosa and in quinin amaurosis. Normal veins and contracted arteries are found in cases of old glaucoma, and occasionally after embolism of the central artery.

Vessels are sometimes narrowed by contraction of their muscular coats, due to some disturbance in the sympathetic nervous system. This may be observed in cases of chronic alcoholism, ergot or quinin poisoning, and preceding fainting.

The vessels may be compressed in the lamina cribrosa; the arteries are contracted and the veins engorged. This is observed in papilledema, albuminuric retinitis, extra-ocular tumors of the orbit, accessory sinus disease, and in some cases of increased intracranial pressure. The two eyes may not be alike; it is important to examine both. Following severe hemorrhages the arteries are incompletely filled and therefore narrow.

Embolism causes the occluded artery to vanish; in addition, the part of the retina normally supplied by that artery quickly becomes edematous and cloudy. Vision is promptly diminished and soon abolished. Thrombosis of a vein produces a great dilatation of the occluded vessel, and large hemorrhages soon follow. In this condition also vision is diminished, but not abolished.

The walls of the veins are flaccid and broadly flattened in some cases of

leukocythemia; the blood column is very pale from the excess of white blood-cells. In lipemia retinalis, observed in some cases of diabetes, the veins are broad and pale; the pallor is due to fat in the blood stream.

In cyanosis retinæ all vessels look as if they were filled with some dark substance, and the fundus is extremely dusky.

White stripes are occasionally seen along each side of a vessel. These may be due to perivasculitis or tissue thickening of the vessel walls, or they may be due to white blood-cells in the adventitia of the walls.

In the region of the disk the vessels may partly or completely disappear in cases of papilledema, optic neuritis, neuroretinitis, and medullated nerve-fibers. This is seldom true in *pseudoneuritis.*

GENERAL DISCUSSION OF RETINITIS

Retinitis, as a term, signifies an inflammation of the retina, which ordinarily it is not. Retinitis is a sign of disease outside the retina in the majority of cases. It may afford the first sign of nephritis, diabetes, cardiovascular disease, syphilis, anemia and leukemia; with such etiology it is expected to appear in both eyes, although it may not do so in all cases. When due to contusions of the eyeball it will affect the injured eye only.

The choroid is often implicated in retinitis, hence the terms retinochoroiditis and chorioretinitis. For that reason it is desirable to consider retinitis according to the situation of the changes in the retinal layers. A good guide to the location of abnormalities is their position with reference to the retinal vessels. The vessels may be in front of the lesion, as in albuminuric retinitis; they may lie behind it, as in some of the pigmented areas of retinitis pigmentosa. The difference in level between the vessels and the lesion can sometimes be estimated by parallactic displacement; the greater the displacement, the greater is the distance between the two.

A study of hemorrhages illustrates the various levels at which a lesion may be found. A preretinal hemorrhage consists of a lake of blood between the retina and vitreous, usually in the vicinity of the macula. It may or may not lie exactly in front of the macula. It has the shape of a pocket; the straight side is always uppermost, and the rounded outline is below. As the patient changes position, the accumulated blood changes location according to gravity. The cause of this hemorrhage is not known. The treatment is rest in bed, potassium iodid, pressure dressings if desired, smoked glasses and visual rest. The general health requires attention.

In general, after excluding mechanical causes such as injury and high myopia, hemorrhages in the retina constitute signs of general diseases. Glau-

coma ought also to be excluded, but it will probably be recognized before hemorrhages occur. A severe hemorrhage may cause glaucoma or hasten its onset.

The periphery of the fundus must be searched for lesions. It is usually necessary to dilate the pupil. A mydriatic can precipitate an attack of acute glaucoma; atropin must never be used for this purpose because it cannot be controlled. Ordinarily a drop of 4 per cent cocain solution and a drop or two of 2 per cent homatropin solution will dilate a pupil in about twenty minutes. As soon as the examination is completed a drop of eserin 0.25 per cent solution or of pilocarpin 1 per cent solution must be instilled.

Linear hemorrhages, which parallel the course of a vessel for any part of its length, and striated or flame-shaped hemorrhages, which look like brush marks, lie in the nerve-fiber layer. The striations tend to follow the course of the nerve-fibers. These are common in kidney disease and in arteriosclerosis. Blood apparently effused into the perivascular sheaths is indicative of infective endocarditis. Hemorrhages that have round or irregular outlines, such as are found in diabetic retinitis, are more deeply situated.

Edema of the disk, papilledema, or choked disk, occurs from a transudation of fluid into the tissues of the nerve head. It may be due to increased intracranial pressure as from brain tumor or internal hydrocephalus, to nephritis with albuminuria, and to thrombosis of the retinal vein. In each of these conditions the veins are engorged, and hemorrhages occur on the disk and in several layers of the retina.

Arteriosclerosis produces hemorrhages in the retina, when the only other sign of the disease may be high blood-pressure (see Plate IX, Fig. A). Many such patients are subjects for cerebral hemorrhage and the prognosis must be guarded. The prognosis is more favorable if the blood-pressure can be controlled. Focal infections must be removed.

When a hemorrhage occurs in a retina and the cause is not removed, more hemorrhages will probably follow. Some will be bright red while others will be brownish. This difference in color gives a clew to the condition, and to the relative ages of the various lesions, but it has no significance in differentiating arterial from venous hemorrhages.

Extravasated blood is absorbed slowly; occasionally the site will be marked by a creamy or grayish spot, and sometimes a brownish stain will persist from the deposit of blood pigment. Where a hemorrhage undergoes resolution connective tissue will be proliferated (see Plate IX, Fig. B); this latter condition is called proliferating retinitis; it often follows an injury, especially a contusion.

Thrombosis of the central vein is characterized by hemorrhages so numerous and massive and so generally distributed that fundus details are largely ob-

scured. The veins are enlarged, engorged with dark blood and tortuous. The arteries are diminished in size and some of the smaller ones may disappear. Where a branch of the central vein is occluded, the hemorrhages will be found in the vicinity of that branch, and corresponding arteries only will be affected. Hemorrhages due to thrombosis occupy various levels, and are found mostly behind the equator. They are situated near the vessels, and are often found in front of the disk. The prognosis is guarded.

Embolism of the central artery is due to the lodgment of an embolus, or to endarteritis obliterans; it is characterized by sudden blindness and invisibility of the artery. Endarteritis obliterans reduces the lumen slowly, but the total occlusion may occur abruptly. Small striate hemorrhages are common, although edema and clouding of the retina are more important signs. The fovea, being nourished by the choroid, shows through the clouding as a "cherry-red" spot. This condition ordinarily occurs in elderly people and the blindness is generally permanent. The existence of a cilioretinal vessel may save some central vision. Ophthalmoscopic examination must be made in every case of sudden blindness.

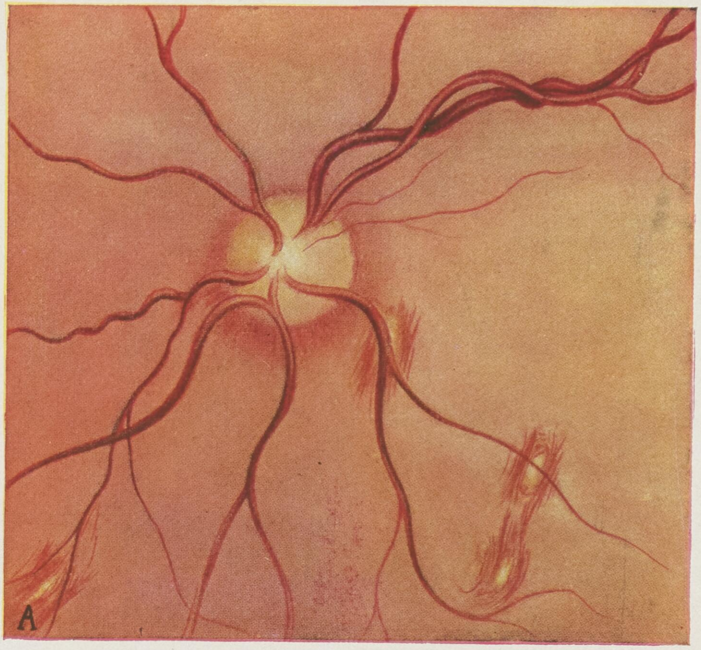
The cherry-red spot is also found in amaurotic family idiocy, which occurs in very young children, mostly of Jewish parentage. It also occurs in quinin poisoning and in commotio retinæ; the history of the drug or of injury should differentiate them.

In embolism of the central artery the patient is usually not seen soon enough for successful treatment. When the patient is attended promptly, gentle and persistent massage of the eyeball serves to restore circulation. Nitroglycerin and amyl nitrite are indicated. Paracentesis of the cornea permits the escape of aqueous and lowers the intra-ocular tension; the vessels expand and the embolus is carried into a smaller vessel where it will do less harm. Later treatment consists of potassium iodid internally, dionin locally, and subconjunctival injections of normal salt solution.

Hole in the macula is due to injury; it is a rare condition and might be mistaken for the cherry-red spot of other conditions. The red area is about two-thirds the size of the disk, and has a punched-out appearance. It has been caused by concussions and head injuries, and may be bilateral.

Recurrent vitreous hemorrhages are rare; they ordinarily occur in young males. The feature of recurrence is a valuable point in differentiation, and the treatment is directed toward preventing the recurrences.

Exudates and edemas are found as white spots in the fundus; they are located in the same manner as hemorrhages. The lesions may be in the retina or in the choroid, or in front of or behind the retinal vessels. Where the spot is in the most superficial layers of the retina or in the vitreous it is in front of



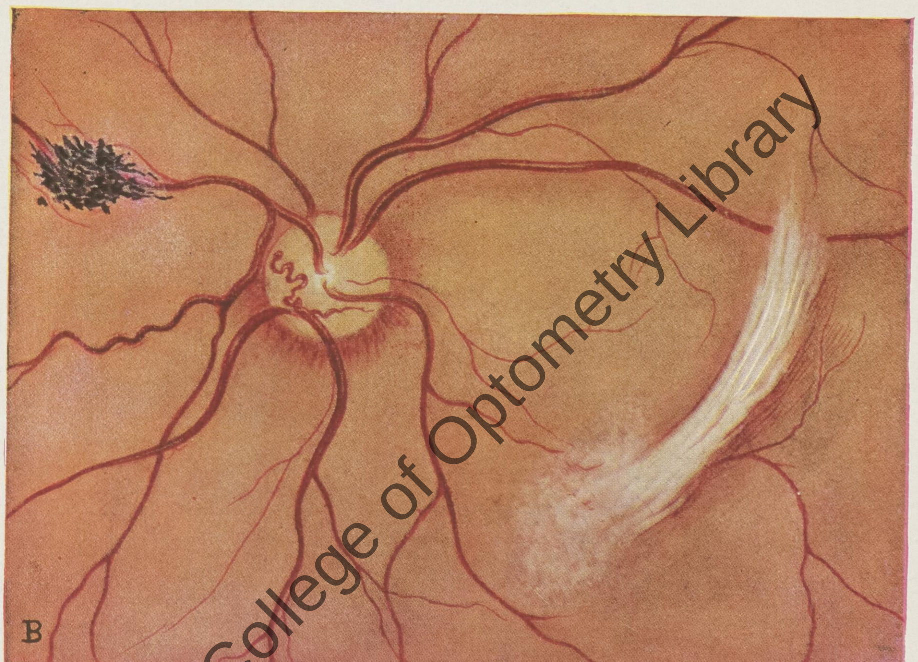


Plate IX.—Retinitis.

A, Retinal hemorrhages and vessel changes; left eye; high blood-pressure.

B, The same eye six weeks later; proliferating retinitis, new vessel on disk and accumulation of retinal pigment. The superior temporal branch of the central artery has been drawn downward. (The right eye is shown in Plate IV, Fig. F.)

a vessel; where behind a vessel it is in the retina or choroid. The association of hemorrhages with white spots is indicative that the latter are in the retina. The association of black pigment with a white spot suggests disease of the choroid, except that a crescent of pigment which borders the disk is without pathological significance.

Choroidal lesions may be hidden by localized edemas of the retina. Such an edema may elevate any retinal vessel that passes over it. These edemas are grayish in color, and are often striated because the nerve-fibers are spread apart by fluid. Strictly choroidal exudates are usually yellowish, while those of the retina are more inclined to be dull pearly white.

Colloid excrescences of the lamina vitrea of the choroid are seen as grayish spots which resemble very small round points of retinal edema. They are usually elevated, and it is often possible to see a faint shadow on one side of them. These excrescences are sometimes accompanied by little accumulations of pigment, and may be mistaken for choroiditis. They are generally multiple, never confluent, and do not destroy the choroid.

Medullated nerve-fibers are anomalies which may be studied in the rabbit's eyes; they have no pathological significance. They arise from the disk or near it, and radiate in patches. They end in a flame-shaped or feathery border, and have been compared to the end of the tail of a white horse. The central vessels or their larger branches wind in and out among the nerve-fibers. The absence of all evidence of pathology is diagnostic. Varicose thickenings of nerve-fibers may very closely resemble medullated fibers, but they will be accompanied by lesions. Both are superficial.

Connective tissue proliferations can occur in any retinal layer. The striations have no necessary relationship to the direction of nerve-fibers. The condition is classed as retinitis proliferans.

Fibrous exudates often resemble localized edemas. They have the appearance of flattened balls of dry cotton.

The white spots seen in albuminuric retinitis are thought to be due to fatty degeneration. They are usually situated in the external reticular layer or along Henle's layer, and seem to attack the supporting fibers of Müller surrounding the fovea. They are arranged radially as a "star figure." Somewhat similar patches are seen in diabetic retinitis.

Larger rounded or oval white spots are often found irregularly surrounding the disk. They have been observed in albuminuric retinitis, diabetic retinitis, neuroretinitis, papilledema from intracranial tumor, embolism of the central artery, thrombosis of the central vein, arteriosclerosis and in some leukemias. The nephritis of pregnancy is occasionally accompanied by the star figure or by the spots around the disk, or by both.

As a rule, all these spots and hemorrhages are associated, regardless of the underlying disease. High blood-pressure is fairly constant. The urine should be examined for albumin, urea and sugar. A general examination should be made when hemorrhages or white spots are found in the fundus.

In septicemia white spots are sometimes found around the disk, but not in the vicinity of the macula. They are accompanied by rounded hemorrhages which have no relationship to the course of the blood-vessels; when the spots are large they may conceal the vessels.

SPECIAL FORMS OF RETINITIS

Albuminuric Retinitis is one of the most characteristic manifestations found in the retina. It is caused by nephritis of any variety, but more especially by the chronic form. Many times the disease is detected by an examination of the eyes. The patient is unaware of constitutional disturbance, but seeks medical advice for failing vision. This fundus picture occurs also in pregnancy nephritis, and in the nephritis associated with scarlet fever and trench fever. The blood-pressure is practically always raised.

Vision is reduced in different ways: The patient often has misty vision as though looking through a veil; scotomata in the visual fields correspond to the retinal lesions; the blind-spot may be enlarged; where the macular region is affected with derangement of the normal relations of the cones, metamorphopsia will be noticed, or a central scotoma may be present. There is no pain, although bright lights may cause discomfort.

Ophthalmoscopic examination occasionally discloses vitreous opacities. There is some cloudiness of the retina with obscuration of the disk, especially of its margins. The disk may be markedly swollen, and closely resemble papilledema. The arteries may be normal or attenuated. The veins are engorged, tortuous and dark colored. Flame-shaped hemorrhages occur near the veins.

White spots are found in two locations in typical cases. Large oval or rounded exudates are situated about the disk, and radiating spokes or white lines, the "star figure," surround the macula. The spots are behind the retinal vessels. The areas about the disk tend to enlarge and become confluent; those about the macula seldom do so. As the white spots develop the vascular changes subside.

The course is slow and the prognosis is bad. Rarely the retinal lesions clear up with restoration of normal vision; they usually undergo slow atrophy. Retinal and optic atrophy, attenuation or contraction of the vessels, disturbance of the retinal pigment and blindness are not uncommon terminations.

Treatment is directed to the cause. Milk diet and rest are indicated. In the nephritis of pregnancy artificial termination of gestation should be considered where the nephritis and retinitis do not react promptly and favorably to treatment. While the prognosis is better in the nephritis due to pregnancy, vision is often damaged permanently, and the terminal stages may leave the patient blind. Dark glasses are restful.

Stellate Retinitis or pseudonephritic retinitis (Duane) may be associated with papilledema from brain tumor, meningitis or internal hydrocephalus, with neuroretinitis, syphilitic retinitis, arteriosclerosis, chlorosis, leukemia, diabetes, influenza, embolism of the central artery and thrombosis of the central vein. With the exception of choked disk due to intracranial disease, in which large white spots are often found around the disk, the principal resemblance to albuminuric retinitis is found in the white spots in the macular region.

Diabetic Retinitis may be indistinguishable from mild or atypical cases of albuminuric retinitis. Small waxy white spots are distributed without regularity around the macular region, and are more noticeable in the vicinity of blood-vessels. Deep hemorrhages are often found between the white spots. The unaffected parts of the retina and the disk usually seem to be normal. The retinal changes sometimes constitute the first demonstrable sign of diabetes. As this disease and cardiovascular-renal disease are frequently associated, the retinal pictures may be very confusing.

Differential Characteristics *

Albuminuric Retinitis	*Diabetic Retinitis*
1. At first a group of bright bluish-white spots is present in the center of the retina, often forming a stellate patch about the macula.	1. Groups of bright glancing spots in the retina, irregular in outline, usually in the central part, but frequently affecting the whole of the fundus.
2. The spots may run together and involve all of the central part of the retina.	2. If the spots are large there still exist small dots and lines, and they never run together.
3. The arteries are narrowed; the veins are large and irregular.	3. The arteries and veins are not much changed in appearance.
4. The optic nerve is swollen and its outline is indistinct.	4. The optic nerve is either not affected or is atrophic.
5. The retina is infiltrated.	5. The retina is not diffusely affected.

The *American Encyclopedia of Ophthalmology* classifies five varieties of retinal manifestations in diabetes. "(1) In central punctate diabetic retinitis there are spots of various sizes and shapes that do not coalesce; small hemorrhages appear between them. (2) In hemorrhagic diabetic retinitis there are

* Oscar Dodd.

hemorrhages but no white spots; it often terminates in hemorrhagic glaucoma. (3) Diabetic albuminuric retinitis has the fundus picture of albuminuric retinitis with the general symptoms of diabetes. (4) Albuminuric retinitis in diabetic subjects is characterized by retinal edema, papilledema, and the vascular changes of albuminuric retinitis combined with the white spots of diabetic retinitis. (5) Atypical diabetic retinitis appears with pigment changes, contraction of the visual fields and night blindness; diabetes is probably an incidental development." This last form suggests the occurrence of diabetes in a patient who has retinitis pigmentosa. Lipemia retinalis should be added to the classification.

Diabetes may be complicated by cataract and refractive changes. Myopia is a frequent development. Rapid reduction of blood sugar occasionally leads to increasing hyperopia. Myopia and hyperopia probably depend upon changes in the lens. Transient peripheral or central amblyopia may occur, and this is thought to be due to changes in the optic nerve which are caused by hemorrhages or toxic substances.

Lipemia Retinalis is an occasional and transient complication of diabetes, especially in younger subjects. The arteries and veins look much alike; they are broad and flat, and very pale or even grayish on an orange background. The blood contains much lipoid materials, which is responsible for the appearances in the fundus. Hemorrhages are uncommon. The lipemia usually clears up after a few days of treatment for the diabetes.

Retinitis Circinata might be mistaken for diabetic retinitis. The white spots are arranged in a wide horseshoe around the macular region. The open end of the shoe points toward and sometimes includes the disk. The spots may be discrete or confluent. Central vision is reduced. No other evidence of pathology appears. The etiology is unknown, the condition is permanent, and no treatment is advised. In complicated cases the macula may be covered by a nebulous cloud, with or without hemorrhages; between this area and the horseshoe the retina seems normal.

An identical horseshoe distribution of exudates has been observed in the choroid. In all doubtful cases foci of infection should be sought for; dionin locally and iodids internally will aid in the diagnosis, particularly if vision is improved after several weeks of treatment.

Syphilitic Retinitis, chorioretinitis or retinochoroiditis is found in both acquired and inherited syphilis. It may appear in one or both eyes. Very often the vitreous is full of fine dustlike particles which some writers regard as pathognomonic. This cloud obscures the retinal details to some extent. The disk is mildly hyperemic; its margins are veiled, but this opacity tends to fade out toward the equator.

Fine pigment spots are mingled with minute white points of atrophy; the retina looks as if it had been sprinkled with "pepper and salt," and this appearance is characteristic of the inherited type. Occasionally new blood-vessel loops arise from near the disk and extend into the vitreous (see Plate IX, Fig. B). The vessels offer little evidence of abnormality otherwise, and hemorrhages are uncommon.

Vision is diminished in most cases, particularly at night. The light sense is reduced more than the visual sense, and this reduction may precede any ophthalmoscopic signs. When an edema in the choroid disturbs the normal relations of the cones in the macula, objects appear to be distorted or diminished in size. Subjective sensations of light and colors may be experienced. Various scotomata often appear in the visual field.

The course of the disease is slow and relapses are common. Progressive changes lead to disseminated choroiditis. Vision should be kept at as near normal as possible with correcting lenses, and dark glasses should be worn. Treatment must be directed against syphilis.

Leukemic Retinitis appears in some cases of leukocythemia of splenic or myelogenous origin, but seldom in lymphatic leukemia; the appearance depends upon altered blood and the excess of white cells. The choroid is pale and the fundus has a yellowish or light orange color. The retina is edematous and the disk margins are indistinct. The retinal vessels are flat and the blood columns are pale; the veins are wider in this condition than in any other. Characteristic oval hemorrhages have creamy centers of white blood-cells and borders of red corpuscles; the same type of extravasations has been observed in pernicious anemia.

Septic Retinitis and Metastatic Retinitis occur in septicemia. The process is probably due to bacteriemia with lodgment of bacterial emboli in the fine retinal vessels. Linear and flame-shaped hemorrhages are common. The tissues of the disk are infiltrated with pus-cells and the picture resembles optic neuritis. Opacities appear in the vitreous. Metastatic choroiditis supervenes and leads to blindness.

Exudative Retinitis or Coats' disease is characterized by large masses of exudates in the retina; these often raise the retinal vessels and resemble tumors. Hemorrhages are common in the vicinity of a mass. The exudates evidently tend to undergo resolution, since detachment of the retina is prone to occur. The course is very chronic. No specific treatment is known. Iodids should be given over a long period.

Retinal Angiomatosis or von Hippel's disease consists of the formation of multiple capillary tumors, in each of which widely distended and tortuous vessels end. The tumors are usually situated peripherally, and continue to

grow in size. Hemorrhages and white spots are associated. No treatment has been of benefit; the disease usually ends in detached retina or glaucoma.

Retinitis Proliferans is thought to be due to traumatic or repeated spontaneous hemorrhages. Organization occurs by a proliferation of connective tissue and the formation of fibrous bands, with or without newly formed blood-vessels, extending from a retinal vessel into the vitreous. Years may be required to develop the band or it may form in a few weeks (see Plate IX, Fig. B). If the band contracts it will probably detach the retina. Subsequent hemorrhages are often followed by similar proliferations. The prognosis for vision is grave. The treatment should be directed toward preventing the recurrences of hemorrhage.

Angioid Streaks in the Retina present a brownish pigmented anastomosing pattern somewhat resembling the arrangement of the large vessels of the choroid. These streaks are believed to result from hemorrhages in the deep layers of the retina with degenerative obliteration of the vessels, or from the formation of new vessels extending into scar tissue. As they are found near the disk, they may be due to changes that occur in ciliary arteries and their branches. No treatment.

Retinitis Pigmentosa is a degenerative process characterized by accumulations of retinal pigment. It can be confused with choroiditis. The disease is bilateral and appears during childhood. It has been observed in several members of the same family, in succeeding generations, and among children of consanguineous parents. Many deaf-mutes have it, and many who have this disease are deaf. Other stigmata of degeneration may coexist. The cause is unknown.

The degenerative process usually begins in or near the equator of the globe. The anterior and posterior uveal blood supplies meet in this region. The capillary layer of the choroid is affected first, and soon the retinal pigment is disturbed. In early stages the pigmentation is limited to the equatorial region. The migration of retinal pigment exposes the vessels of the choroid and the fundus becomes "tessellated." Degeneration of the retinal nerve-fibers leads to consecutive optic atrophy; the disk is waxy or yellowish in color. The retinal vessels are very fine, few in number, and almost invisible in the periphery of the fundus.

The most characteristic feature is the distribution of the pigment; it follows the retinal vessels, especially the veins, and lies mostly in front of them. The pigment inclines to brown; it accumulates in stellate or branched figures that have been compared to bone-corpuscles. When the figures lie closely together their processes join, and the pattern resembles a system of haversian canals. There are no exudates, white spots or hemorrhages.

In disseminated choroiditis the pigment is black, and it accumulates without a regular arrangement mostly or entirely behind the retinal vessels; the latter are not materially affected.

Symptoms.—The earliest manifestation of the disease is night-blindness. Progressive concentric contraction of the visual fields results in tubular vision. When central vision is lost before the contractions of the field are complete, the patient has sight only from a zone surrounding the macula. When this zone is the first part affected, the patient has an annular scotoma. Total loss of vision often occurs in the sixth decade of life.

Bilateral cataracts generally form after the fields are well contracted. If the cataract is central it will be necessary to dilate the pupils to map the visual fields and examine the fundi. In this disease poor vision in reduced light is not improved by dilating the pupil. Nystagmus is a common feature.

Treatment.—There is no specific. Strychnin has seemed to do good when given in large doses hypodermatically over a long period of time without intermission. It is less effective when given by mouth. Galvanism and thyroid extract have been advised. Vision must be maintained at its maximum with correcting lenses. Cataracts are to be removed, when indicated, by combined extraction. Some investigators regard retinitis pigmentosa as an expression of syphilis, but antisyphilitic treatment is without effect. The fields must be mapped with frequent regularity to follow the effects of treatment.

Retinitis Pigmentosa sine Pigment has all of the signs and symptoms of the pigmented form except the pigment, and that may appear in due time.

Retinitis Punctata Albescens is allied to the preceding forms. Innumerable white points are distributed over the whole fundus except in the macular region. Night-blindness and some contraction of the visual fields are the principal symptoms. It is not progressive, there is no atrophy of the optic disk, and the retinal vessels are normal.

Congenital Night-Blindness is stationary. It is often found in several members of the same family. The vision is good except in reduced light. Other evidences of ocular disease are absent.

A **Cherry-red Spot** in the macula is observed in embolism of the central artery, amaurotic family idiocy, quinin poisoning and commotio retinæ. The first two have been mentioned. Amaurotic family idiocy is not to be confused with amaurotic family dementia or familial maculocerebral degeneration, which occurs in older patients and is not confined to the Jewish race; both are rare.

De Schweinitz illustrates an instance of a cherry spot in an area of edema at the macula in a case of syphilitic chorioretinitis with sclerotic arteries. The author observed a small brown spot, diagnosed as a hemorrhage, in the foveal area in a case of renal retinitis with hemorrhages and numerous large exudates

in a patient at the Coleman Hospital, Indiana University School of Medicine.

Quinin Poisoning is characterized by extreme attenuation of the retinal vessels, only a few of which are visible. The retina is pallid from ischemia. The pupils are dilated and immobile in early stages. The cornea is often insensitive. The visual fields are contracted and night-blindness is common, although light and color senses may be preserved. Tinnitus and deafness are usually associated with it. The treatment is to raise arterial tension.

Commotio Retinæ or Berlin's opacity occurs at the site of an injury, usually a contusion, or opposite the site. Direct contact with the eyeball is unnecessary; an explosion concussion or a fall on the head may cause it. A soft or milk-white diffuse cloudiness is found, with a red spot over the fovea. It has the appearance of an edema. The cloud disappears spontaneously after a few days and vision is restored gradually. A common feature of retinitis is found in the tortuosity of the fine vessels. The eyes should be protected with dark glasses.

Solar Retinitis is caused by looking directly at the sun, particularly during a partial eclipse, without eye protection. A dark spot appears in the central field of vision, and this persists according to the duration of the exposure. The fovea may show a change of color by early pallor and late darkening, or the foveal reflex may be changed. The ophthalmoscope reveals little else. The treatment is visual rest and dark glasses, strychnin, and subconjunctival injections of normal salt solution.

Electric Retinitis may produce the same signs as solar retinitis. More intense effects are produced by exposure to arc lights or to electric welding light. The symptoms resemble those due to snow-blindness. Pain is usually a prominent symptom; it is probably due to exfoliation of the corneal epithelium and spasm of the pupillary sphincter. The lids are red and swollen, and a mucous secretion is frequently present.

The irritation persists for several days or weeks, and is usually followed by full restoration of vision. Dark glasses are needed. Cold applications are useful during the height of the irritation. Atropin is needed to relax the pupil, and phenacain ointment is soothing for the corneal irritation.

Niphablepsia or snow-blindness affects the conjunctiva more than the retina, so far as objective evidence is concerned. Blepharospasm, lacrimation and sensations of the presence of a foreign body are prominent. Photophobia directs attention to corneal injury. The pupil is contracted. Retinal exhaustion may account for the blindness. The effects pass off in a few days. The treatment is the same as for electric retinitis.

DETACHMENT OF THE RETINA

Detachment of the retina is more accurately a separation between the pigment layer and the layer of rods and cones. The separation may come on gradually or abruptly.

The patient complains of obscured vision in part or all of the field of the affected eye, of looking through a dark cloud or curtain, of seeing flashes of fire or light, of seeing objects distorted in outline or diminished in size, of seeing only parts of objects, of seeing the object doubled, monocular diplopia, or of disturbances in the light and color senses. Central vision will be retained while the macular region is intact and no folds of retina come between it and the pupil.

The external appearances of the eye are normal. Intra-ocular pressure is lowered. The anterior chamber will be deep where the lens has been drawn backward, and the iris may tremble with eye or head movements. In late stages the lens may be cataractous.

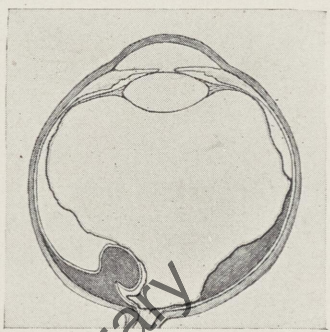


FIG. 145.—TUMORS OF THE CHOROID.

The detachment is hard to find when it is small and flat, and when the vitreous or lens contains opacities. The visual fields should be plotted with different sizes of blue test objects as well as with white ones. Scotomata are searched for with small objects, 1 or 2 millimeters in diameter. The patient must be able to maintain fixation or satisfactory field work cannot be done.

Retinal detachment is due to underlying fluid or to tumor. The first is a true detachment and the latter is an elevation. In the first the intra-ocular tension is usually below normal, while it is above in the latter. When a tumor is in the choroid all layers of the retina are elevated, but where it is in the retina the pigment layer usually clings to the choroid. Detachment of the retina is a complication of intra-ocular tumors (Fig. 145); it is necessary to exclude tumor in all cases of detachment. By transillumination a shadow is formed in most cases of tumor, while in simple detachment the light will be diffused but clear and reddish. Flat unpigmented tumors obstruct the light very little or not at all.

The pupil should be dilated. Oblique illumination will often reveal an anterior detachment. In making the direct ophthalmoscopic examination it is best to begin with the open aperture at a distance of 75 centimeters (30 inches), and gradually approach to 15 centimeters (6 inches), using lenses from plus 1 to plus 7. While doing this the patient rotates the eye in various directions.

The fundus reflex is red when normal; it is gray from detached portions of retina. The retinal vessels will be seen at different levels.

The indirect method brings into view much larger areas of fundus, and details are shown in sharper contrast by it. By moving the magnifying glass before the eye, all parts of the retina may be inspected.

The detached retina is a translucent gray lusterless membrane arranged in billowy folds which undulate or tremble with eye movements. The crests of the folds reflect a whitish light, while the intervening portions absorb light. The retinal vessels over a detachment are chocolate in color and have no central light streak. They follow the folds and have a semblance of tortuosity; they disappear and reappear as the surface of the retina is concealed or visible. It is best to focus on the vessels when examining the folds. The disk is sometimes concealed by the detachment.

Flat detachments resemble edemas, but the chocolate color of the vessels helps to distinguish the detachment. When an edema of the retina overlies a choroidal lesion which does not reflect light, the vessel crossing it is red and has a light streak. A flat detachment or elevation of the retina may conceal a choroidal tumor in its early stages.

Vitreous opacities are common in retinal detachment. Choroiditis with pigment accumulations and atrophic areas are not uncommon. Hemorrhages and exudates may be present. When the retina tears or ruptures, the normal or diseased choroid may show through the rent. Changes belonging to myopia are occasionally found.

When a tumor elevates the retina the surface is smooth or nodular, and does not lie in folds; there is no tremor or undulation with eye movements. The accumulation of fluids due to the presence of a tumor will in time detach the retina, and then folds will be formed. This feature emphasizes the necessity for transillumination.

Detachment of the serous type usually occurs above first. As the fluid settles down the detachment will be transferred to the lower part of the eye, and the retina may or may not reattach above.

When the detachment is complete, the retina preserves its bonds only at the disk and the ora serrata. Viewed from the pupil it forms a fluted funnel; in longitudinal cross section it is trumpet shaped. Connective tissue proliferation and contraction continue until the retina is represented by a cord that extends from the disk to a point behind the lens; from this point the membrane flares out to the ora serrata. Complete detachment is not discoverable by the ophthalmoscope; in this condition the lens quickly becomes opaque.

Detachment of the retina is due to myopia, recent or old injuries, hemorrhages or exudates in the choroid, uveitis, shrinking or loss of the vitreous by

diseases or wounds, and to tumors of the choroid and retina. The retina may be pushed off the choroid by subretinal fluids or growths, or pulled off by the contraction of new connective tissue which extends from one part of the retina to another. Detachment may be an accidental complication in operations for cataract or glaucoma, when intra-ocular tension is lowered too abruptly.

Treatment of simple detachment is directed toward absorbing the fluid and securing reattachment of the retina. Diaphoretics and iodids are indicated. Subconjunctival injections of normal salt solution, or solutions of 4 to 8 per cent (Swanzy) should be used. Dionin and atropin are used locally. Several weeks in the dorsal position might encourage the retina to settle into place and become reattached. Few patients will submit to this regulation, and it might prove disastrous to life in an aged person. An elastic bandage is recommended by some surgeons.

Operative measures attempt to drain the fluid from beneath the retina. This may be done direct by a scleral window protected with conjunctiva, or by withdrawing the fluid through a hypodermic needle. Less effective is the plan of forcing the retina back by filling the vitreous chamber with an artificial vitreous.

Prognosis is bad because of progression or recurrences; it is a chronic condition. Cataract, softening of the globe and atrophy are the usual terminations.

Detachment caused by a neoplasm calls for immediate enucleation of the globe.

CHAPTER XI

INTRA-OCULAR TUMORS

Sarcomata of uveal tissues in adults, and gliomata of the retina in infants and young children are the principal tumors that originate within the eyeball. Many other neoplasms have been reported, but they occur very rarely. Of sarcomata, those involving the choroid are the most common.

SARCOMA OF THE UVEAL TRACT

Sarcoma of the Iris begins as a nodule and grows rapidly. It may perforate the coats. Metastasis often occurs. This tumor has been removed by excising the affected section of iris by iridectomy. Such a specimen should be submitted for laboratory diagnosis promptly and invariably; if malignant, it is advisable to enucleate the eye, which some authorities advise should be done in the first place. Melanomata resemble iris sarcomata; they are benign and of slow growth. Other growths of the iris are cysts, gummata, and tubercles.

Sarcoma of the Ciliary Body is usually seen as a dark or yellowish crescent at the root of the iris (see Fig. 140). In this situation it resembles iridodialysis. Light passes through the latter but the tumor is opaque. A cyclodialysis must be excluded, usually by a history of injury. Blood-vessels and pigment are sometimes seen on the surface of the tumor. The lens may be displaced and the ciliary muscle may be inactive at the site of the tumor. The anterior ciliary vessels are generally engorged in the segment corresponding to the location of the tumor. In time extension will involve the choroid, or the tumor may grow outward. Enucleation is advised.

Sarcoma of the Choroid originates in the outer layers of the choroid. It is primary, single, and occurs in but one eye. It is mostly observed in the cancer age between forty and sixty. The disease rarely occurs in children; statistics on thirty-one cases ranging in age from one and one-half to fifteen years have been reported. It is called melanotic sarcoma or leukosarcoma, according to whether it is pigmented or not; the pigmented variety is far more common.

The growth consists of round or spindle cells or both. Visual defects depend upon the location and extent of the tumor. When in the macular

region central vision is diminished or lost; where well forward and below, vision is little affected. It generally causes a scotoma in the visual field.

Choroidal sarcomata present a sequence of symptoms that are divided into four stages. First, the quiet stage of growth without symptoms other than perhaps visual defects. Second, the stage of secondary glaucoma or of iridocyclitis. Third, the stage of rupture outside the globe. Fourth, the stage of metastasis.

In the first stage, a yellowish brown or dark circumscribed flat mass is seen ophthalmoscopically. The course of the retinal vessels crossing it will demonstrate any elevation of the retina. Later on the retina is detached by a serous effusion or by an albuminous liquid. The margins of the tumor are then poorly defined. The further accumulation of liquid produces a true detachment of the retina; it lies in folds about the tumor or to one side of it, or below it if the tumor is high up in the eye.

When the retina detaches, vision is affected and the patient seeks advice. This is often the first occasion for examination, and the tumor may be concealed by the folded retina by this time. Transillumination with a dilated pupil is required. Where the tumor lies in front of the equator this procedure is not difficult with one of the usual instruments; when it lies behind, the transilluminator devised by Lancaster is of service. It is said that negro eyes, being densely pigmented, are difficult to transilluminate satisfactorily. While the instrument is moved about over the detachment the pupil glow is observed. The glow is diminished by a tumor. The shadow is dense according to the degree of pigmentation.

Where the retina is not detached or is transparent, new blood-vessels and pigment patches which belong to the tumor may be seen with the ophthalmoscope.

The tumor grows from the outer layers of the choroid toward the center of the eye. It may grow through the layers of the choroid, which are eroded by it, or it may rupture the lamina vitrea and expand after it has gained a position inside that membrane. In the first instance the tumor is sessile, while in the second it is pedunculated (see Fig. 145). These are laboratory distinctions; the clinical features are seldom clear.

In time the detached retina affects the lens and it becomes cataractous. No details can be seen through it, but it does not obstruct transillumination; some light is always transmitted except in the presence of blood or pigmented tumors. Leukosarcomata will transilluminate (Swanzy).

Duane suggested thrusting a fine sterile needle into the suspected tumor mass. If the needle is fixed, tumor is present; when it can be freely moved, the retina is detached. All cases of detached retina should be searched for

tumor, especially *when the intra-ocular pressure is raised.* If choroidal vessels can be seen at the suspected site, tumor is improbable. The retina does not usually rupture when detached by a tumor; there are no windows through which the choroid can be seen. Ruptures in the retina constitute a differential point against the diagnosis of tumor.

In the *second stage,* progression of the growth seldom excites an iridocyclitis. The common complication is secondary glaucoma with the usual train of symptoms: Increased tension, pain, engorged conjunctival and anterior ciliary vessels, hazy media, and diminished vision. The lens and iris may be crowded forward and reduce the depth of the anterior chamber. The hazy media interfere with an ophthalmoscopic examination.

Where there is no history of the prodromals of glaucoma, such as limitation of the nasal visual field, pain, or diminished vision, and transillumination demonstrates a shadow, tumor may be assumed to exist primarily and glaucoma secondarily. All cases of glaucoma should be examined for tumor. Detached retina with increased tension means tumor in nearly every case. The lymphatic glands are seldom enlarged up to this time.

The *third stage* is marked by rupture through the sclera along the anterior ciliary vessels or along the vortex veins near the equator; rupture seldom occurs along the posterior ciliary vessels or the optic nerve. The tissues break down, and the ocular, orbital and palpebral structures are subject to invasion by the malignant process. The *fourth stage* of metastasis is ordinarily to the liver, but it may involve other structures.

Treatment is to enucleate early, or as soon as the diagnosis is reasonably certain. When this is done in the first stage metastasis may be avoided. There is much less assurance in the second stage. Third stage sarcoma demands exenteration of the orbit, with removal of such bones as show suspicious characteristics. The operation should be followed by deep x-ray or radium treatment. Although it is not known that intra-ocular sarcoma extends by way of the optic nerve or of its sheaths, it is the rule to include as much as possible of the nerve when enucleating.

Ring Sarcoma of the ciliary body follows the circumference of that body. It is probably an infiltration of sarcoma cells. *Flat sarcoma* of the choroid is also an infiltration. The choroid is evenly thickened, and the retina overlies it as a shallow detachment or elevation. Both are thought to spread along the lymphatics. These may be endotheliomata (Parsons).

Secondary Carcinoma of the Choroid is usually bilateral, and most frequently is incidental to cancer of the breast. It is nonpigmented. It spreads evenly and widely, and the retinal detachment is shallow. The detachment and impaired vision may constitute the only evidence of ocular involvement;

the history or knowledge of the primary disease should fix the diagnosis. It appears late in carcinomatosis.

Melanomata of the Choroid are slightly elevated circumscribed slate-colored areas which have uniformly striated flat surfaces. They are benign. The striations are in the direction of the nerve-fibers overlying the edema.

Detachment of the Choroid exhibits a dark mass in the vitreous. The retina is elevated with it, but not from it necessarily. Where the retina is transparent, the stroma of the choroid will be visible equally with some of the retinal vessels. The lesion may be multiple. The anterior chamber is shallow and the media are not clear. It has followed operations for glaucoma or cataract as a late complication. Prognosis is guarded. Mercury is advised.

Intra-ocular Vitreous Hemorrhage excludes examination by ophthalmoscopy and transillumination. The pupil is intensely black and opaque. Duane's needle test may differentiate it from melanotic sarcoma. A history of injury is good evidence for hemorrhage, but a hemorrhage might come from injury to an eyeball that contains an unsuspected tumor. Subconjunctival injections of normal salt, dionin locally, and potassium iodid internally are indicated.

Tubercles of the Choroid appear in miliary form, or as a conglomerate mass. They are yellowish. A large tumor is sometimes associated with outlying small ones. It can rupture through the sclera. Other evidence of tuberculosis may be found. The tuberculin test should be made.

Exudates in the choroid and retina must be excluded in the diagnosis of intra-ocular tumors.

GLIOMA OF THE RETINA.

Glioma of the retina is a nonpigmented malignant growth which begins in any cell layer of the retina. It is primary and multiple. The second eye is affected independently in about 20 per cent of cases. It is a disease of infancy and early childhood, it rarely appears after the age of five years, and it is practically unknown after sixteen. Glioma has been observed in successive generations, and in several members of the same family of children. It is called glioma which it is not, retinoma, retinocytoma, neurocytoma, neuroepithelioma, and retinoblastoma; Jackson urges the last as a discriminating term.

The untreated cases undergo four stages. There are but few signs and symptoms in the first stage. A whitish or yellowish reflex from the pupil with evidence of impaired vision calls attention to the condition. This reflex has been called "amaurotic cat's eye," because the eye is blind and the pupil glows like the eye of a cat in the dark. The pupil is dilated, and if necessary an anesthetic is given, to aid in making a thorough examination.

Glioma grows from the retina inward or outward. When it grows inward it is called glioma endophytum (Fig. 146). A main tumor and many smaller masses may be seen with the ophthalmoscope, and sometimes by oblique illumination. Retinal detachment is absent or delayed in this form. When the tumor grows outward it lies behind a detachment of the retina and is called glioma exophytum (Fig. 147).

In the first stage glioma must be differentiated from a group called "pseudogliomata," because of confusing resemblances. The posterior surface of the lens may be lined with a sheath of persistent fetal blood-vessel remnants; they are not progressive. Suppurative choroiditis, metastatic ophthalmitis, endophthalmitis and abscess of the vitreous are supported by a history of a recent acute febrile disease like meningitis, or an infected injury.

Simple detachment of the retina usually shows the signs of detachment by clear transillumination, movement of the fluid mass, and the absence of new

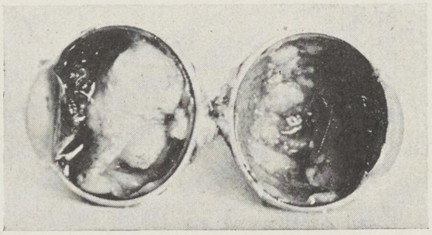


Fig. 146.—Glioma Endophytum.

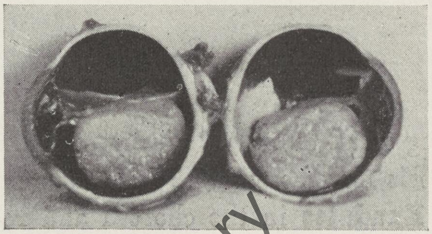


Fig. 147.—Glioma Exophytum.

blood-vessels. These latter are practically always present on the surface of a glioma. Tuberculous masses in the eye of a child may be impossible of differentiation. This should be treated as a glioma, except when tuberculin tests are positive. The differentiation from sarcoma is of laboratory interest only, although age may be considered.

In the second stage the intra-ocular tension is increased. In the pseudogliomata the tension is generally low. When the retina is detached in glioma the tension may be temporarily low. Where the tension is raised, pain is a prominent symptom. The eyeball may appear to be enlarged. The tumor continues to grow until it fills the globe and perforates its walls.

The third stage is marked by rupture of the globe, usually at the limbus. The mass continues to grow into the orbit and outwardly between the lids. It bleeds readily, and has been called "fungus hæmatodes." The fourth stage is characterized by metastasis or direct extension. Neighboring lymph glands, the orbit, brain, bones of the head, and sometimes distant organs are invaded. The second eye is involved independently; the tumor is primary in each eye.

Histologically, the tumor consists of cells having large nuclei and little cytoplasm; there is no true stroma. The round-celled unpigmented tissue is suggestive of sarcoma. The cells form tubules which are blood channels without endothelial linings. These formations are called cell mantles. The rapid proliferation of cells about the blood column crowds the older cells away until they are no longer sufficiently nourished. The scanty intercellular substance is lost and the older cells fall apart. The cytoplasm disappears and the nuclei disintegrate with a gradual loss of staining power.

This process occurs in areas which later undergo necrosis and liquefaction; calcareous material is deposited in them. Sometimes the "rosette" figure is found; spokelike cells are arranged about the blood column (Fig. 148). The tubular character does not depend upon the formation of rosettes however.

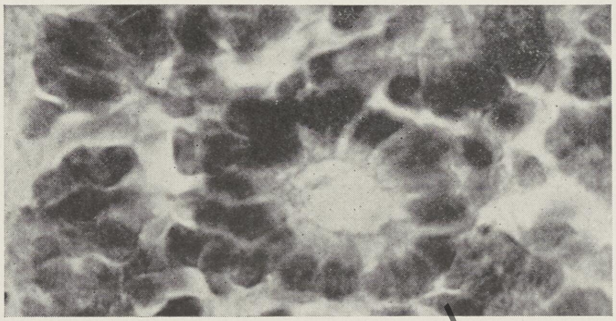


Fig. 148.—Rosette in Glioma.

The tumor cells closely resemble lymphocytes and the nuclear cells of the retina. It is difficult to identify an isolated cell.

In one case of bilateral involvement the right eye was enucleated at the age of four months and the left at seventeen. The first showed an abundance of rosettes, the other none. The first was glioma exophytum and the second was glioma endophytum.

Metastasis depends largely upon the liberation of individual cells and their freedom to migrate anywhere by the lymph stream. A vigorous cell can initiate a new proliferation in any tissue where it may be deposited. The brain may become involved either by direct extension or by metastasis by way of the optic nerve. It is important to take as much of the nerve as possible when enucleating. Direct extension to orbital tissues only occurs after rupture of the eyeball or of the optic nerve sheaths.

Treatment consists of enucleating the globe as soon as the diagnosis is reasonably certain. The optic nerve stump must be examined microscopically, and if extension is apparent the orbit must be exenterated. In bilateral cases one eye is enucleated and subjected to laboratory examination. When the diagnosis of glioma is established, the second eye may be removed. Deep x-ray or radium is advised by some, to be applied to the orbit after the removal of the tumor. Some advocate this treatment for the second or less involved eye. Should the growth show progress under this treatment, the eyeball can be

enucleated later. A few apparent cures by x-ray and radium have been recorded. An eye so treated may shrink. All sightless eyes with a suspicion of glioma should be excised.

Prognosis is one as to life. Where an eye is enucleated within ten weeks after the first appearance of glioma there is a fair prospect for life. A lapse of three years from enucleation without signs of metastasis gives confidence in cure, but the patient should be kept under observation for some time after that.

CHAPTER XII

THE OPTIC NERVE

ESSENTIALS OF THE ANATOMY OF THE NERVE AND DISK

The Nerve.—The essential elements of the optic nerve are the fibers or axis cylinders that arise from the ganglion-cells of the retina. These are gathered into a trunk which leaves the eyeball at the scleral foramen, crosses the orbit, passes through the optic canal, and enters the cranial cavity to terminate at the chiasm; each portion is named from its situation: ocular, orbital, canalicular and intracranial. The orbital portion is doubly curved like an italic *f* (Fig. 149) so that it neither restricts the movements of the eyeball nor is put on a tension by such movements.

The nerve-fibers are gathered into about eight hundred bundles, which are supported by neuroglial fibers. Connective tissue forms septa between the bundles and unites them into a trunk. In structure the optic nerve resembles a column of the spinal cord or a brain tract. It carries sensations of light, visual images and impulses for pupillomotor activity.

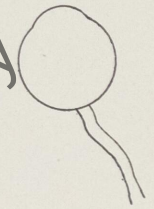


Fig. 149.

Connective tissue and fibers from the choroid and inner layers of the sclera form a sievelike structure, the lamina cribrosa, across the scleral foramen. The nerve-fibers pass out through the meshes of the lamina cribrosa and at once acquire medullary sheaths. Consequently the extra-ocular part of the nerve is thicker (see Plate I).

The outer layers of the sclera blend with the dural sheath of the nerve. The cerebral meninges invest the nerve as a sheath through the optic canal and the orbit. The pia invests the intracranial portion also; it fits the nerve trunk snugly, and its fibers blend with those of the septa to support the nutrient blood-vessels. The dura is loosely fitted to the nerve. The intervaginal space lies between the dura and pia.

The arachnoid divides the intervaginal space into subdural and subarachnoidal spaces. They apparently terminate at the scleral foramen. Endothelial linings suggest that these spaces are lymph channels which are continuous with the cerebrospinal fluid spaces about the brain. Lymph-spaces are found between the bundles of fibers in the nerve.

The blood supply of the nerve is from the anastomotic circle in the ocular portion, and from the vessels of the pia.

In the optic canal the nerve is accompanied only by the ophthalmic artery. The central artery of the retina pierces the sheaths of the nerve on its inferior surface, crosses the intervaginal space, and penetrates the substance of the nerve to the axis; this it follows forward to the porus opticus or physiological cup. The central vein of the retina follows the course of the artery, but leaves the nerve somewhat anterior to it. Both pierce the sheath from 8 to 17 millimeters behind the globe. These vessels occupy perivascular sheaths within the nerve.

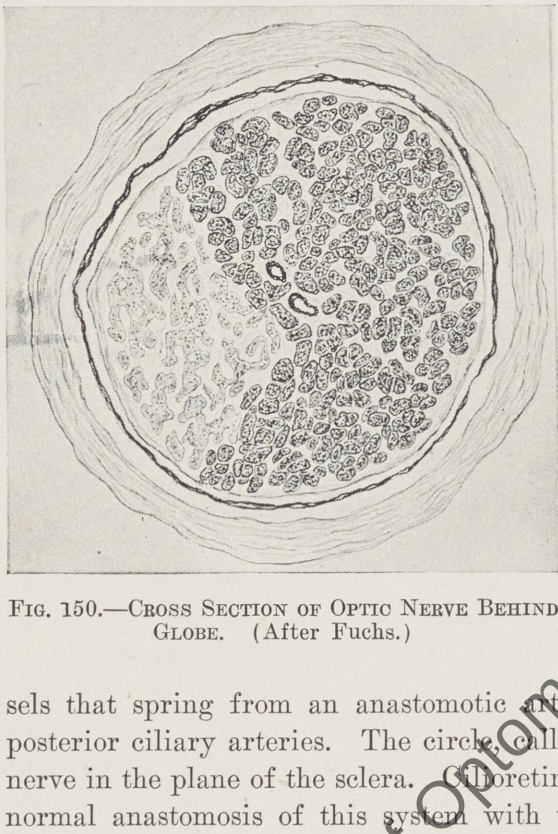


Fig. 150.—Cross Section of Optic Nerve Behind Globe. (After Fuchs.)

The Optic Disk.—The visible portion of the optic nerve is called the nerve-head, papilla or disk. It is prominent in the fundus because of its definite margins, its lighter color, and the convergence of the retinal vessels upon it. The margins are distinct, for neither the retina nor choroid normally quite conceals the narrow border zone of sclera that surrounds the ocular end of the nerve. The color is lighter than the surrounding fundus because pigments are absent. The color is not white, but pink from the numerous fine vessels that spring from an anastomotic arterial circle derived from the short posterior ciliary arteries. The circle, called Haller's or Zinn's, surrounds the nerve in the plane of the sclera. Cilioretinal vessels are given off from it. No normal anastomosis of this system with the retinal vessels has been demonstrated.

The temporal quadrant of the disk is distinctly pale, since it is less freely supplied with capillaries. This section is occupied by the papillomacular bundle, or the fibers from the macular region (Fig. 150). While this section leaves the eye on the temporal side of the nerve trunk, it quickly acquires an axial position which it holds almost to the chiasm.

Disks are rather constantly 1.5 millimeters in actual diameter. Distances in the fundus are estimated in terms of "disk diameters" regardless of ophthalmoscopic variations in nerve-head dimensions in different eyes.

EXAMINATION OF THE DISK

The examination of an optic disk should consider in order the form, size, color, margins, level or plane, and blood-vessels.

The disk is round or oval; under cycloplegia it may change form. In one patient the disk appeared circular in outline; after cycloplegia, which disclosed 3 diopters of hyperopic astigmatism, it was oval. So the form of the disk may be altered in appearance by astigmatism or by segmental contraction of the ciliary muscle. The apparent size of the disk depends upon the refraction. By direct ophthalmoscopy the hyperopic disk appears small, the myopic large; by the indirect method the reverse is true.

The color of the disk is in contrast to the surrounding eye-ground. It will appear pale in a dark fundus, and pink in a pale fundus. Grayish stipplings in the disk or in the physiological excavation represent the apertures in the lamina cribrosa. This is most pronounced in primary or gray atrophy of the optic nerve. A uniformly white or pale yellowish disk results from secondary or white atrophy.

The margins of the disk are definite in normal states, generally sharp in atrophies, and obscured or blurred in edemas and in some other conditions. In the average case the disk is surrounded by a white ring, and this by a black ring. When these rings are incomplete, parts of them appear on the temporal side usually. The white portion is made up of connective tissue or sclera, and the dark of retinal or choroidal pigment. Either or both rings may be absent.

In some myopic eyes the sclera becomes thin and yielding; it bulges backward and forms a posterior staphyloma. The choroid and retina are pulled away from the disk; when this occurs equally in all directions, a peripapillary staphyloma is formed. When it occurs in but one direction, usually temporally and below, a crescent staphyloma, temporal conus or traction crescent is formed. Sometimes the retina and choroid are pulled partly across the disk like a sliding curtain. The traction crescent shows as before, while the retina and choroid overlying the nasal portion of the disk have a yellowish color; this is called a supertraction crescent.

These changes about the disk may make it appear greatly enlarged. The position of the entrance of the central vessels is often significant; they are far to one side and appear to emerge from beneath the edge of the supertraction crescent. Sometimes the retina only is drawn, and the choroid is exposed to view in the situation of the crescent. Other myopic changes will be found in these eyes.

The disk margins are blurred or concealed in neuroretinitis, in optic neuritis, in papilledema, and in some cases of hyperopia in which it is called

pseudoneuritis (see Plate X, Fig. C). Other evidences of congenital anomalies or pathology are usually associated with blurred margins.

The plane of the normal disk is level with the surrounding retina. Occupying the central part of it is a depression called the physiological excavation, the porus opticus, or the vessel funnel. From here the vessels spread over the retina. The depression may be eccentric in cases where the nerve leaves the eyeball at an angle, and it may appear to be so in some refractive states. Sometimes the depression is barely discernible. In this form the vessels make an abrupt bend to follow the nerve-fibers of the retina. According to the depth of the cup the vessels curve gently or sharply in emerging from it.

The vessels offer a guide to the width of the cup, since their bendings mark the edge. Where a cup gradually shelves to the margin of the disk, the vessels slope across it without pronounced bending. A steep excavation at the margin of the disk must be considered pathological. An abrupt bend in a central vessel at the disk margin is significant of glaucoma. The cup may be present but obscured by pial tissue, by tissue from the sheaths of the central vessels, by tissue newly formed following inflammations, or by edema.

In primary atrophies of the optic nerve the disk is depressed, but it slopes gently from the margins. In cross section the floor is saucer-shaped; it is difficult to demonstrate this ophthalmoscopically. The disk is occasionally elevated above the surrounding retina in cases in which its margins are blurred or hidden. In these instances the central vessels may lie on the disk, or they may be only partly visible as they wind in and out of the swollen tissues.

The level of the disk compared to that of the surrounding retina may be measured by the lenses in the ophthalmoscope. In this examination the accommodation of the examiner must be suspended. This can be effected by using the direct method and keeping both eyes open. A retinal vessel near the disk is first brought in focus. Then attention is directed to the disk to estimate its level.

Weaker plus or stronger minus lenses are required to see the bottom of a depression, and stronger plus or weaker minus lenses are required to see the apex of an elevation. One diopter of lens equals approximately one-third millimeter of depression or elevation. Focusing of less than one diopter is accomplished by varying the distance between the observing and observed eyes, and by slight side to side movements by the examiner.

Pallor of the disk accompanies general anemia, or anemia of the retina can be due to contracted or partially obstructed arteries. It may be more apparent than real; the width of the vessels should be noted. The light sense, light difference and visual fields should be tested; early retinal fatigue may be important in diagnosis.

Hyperemia of the disk may also be more apparent than real. Repeated examinations, noting any changes that occur, are helpful in determining stationary conditions or progressive tendencies. Comparisons between the two eyes offer suggestive evidence. The fine vessels on the disk are too plainly seen in true hyperemia. The retinal vessels may be engorged and tortuous. The disk margins may be slightly blurred or hazy in outline. Vitreous haze must be excluded, for this dims the outlines of the disk and suggests a redder tone for its surface.

Hyperemia results from exposure to intense light, from toxic influences and from refractive errors. It may be the forerunner of neuritis or of papilledema.

INFLAMMATIONS OF THE NERVE

The Disk

An inflammation may develop in any portion of the nerve. When this occurs in the disk, it is *optic neuritis;* where a considerable surrounding area of retina is involved, it is *neuroretinitis;* when there is considerable engorgement of the disk with an elevation of 2 diopters or more, it is *papilledema* or choked disk. When the nerve is involved, often without ophthalmoscopic signs but with subjective symptoms such as diminished vision, it is called *retrobulbar neuritis.* One form may blend with another, and retinitis of some degree is usually associated. *Pseudoneuritis* must be excluded.

Optic Neuritis and neuroretinitis are caused by a transudate or an exudate into the tissues of the disk; this extends into the retina for a variable distance, usually as a hazy border whose width varies up to one or two diameters of the disk. Details are obscured by the cloudy exudate, there is an abnormal redness without much elevation, the arteries are unchanged, and the veins are full and tortuous. Hemorrhages are rare. Vision is materially impaired early, and a central scotoma is often present.

The tendency of optic neuritis is toward blindness. When the cause of it is promptly removed the prognosis is better for the preservation of vision, although it may permanently deteriorate later. The inflammatory symptoms subside rather slowly. Postneuritic atrophy of the disk generally follows severe inflammations. The tissues at first have a hard red appearance which gradually changes to white. Connective tissue is proliferated as the exudate is absorbed, and this conceals the details of the disk.

Optic neuritis is often found in cases of orbital or sinus disease, in which it will probably be limited to one eye. When it is bilateral it is suggestive of syphilis, renal disease with albuminuria, and other illnesses in which the general symptomatology will probably attract attention before the visual dis-

turbances are noted. When optic neuritis is due to arteriosclerosis it may resemble a papilledema.

In syphilitic cases opacities are commonly found in the vitreous. These often exist in sufficient amounts to obscure the fundus details, and they add to the evidence for the diagnosis of syphilis. Confirmatory choroidal changes, sclerosis of the retinal vessels, hemorrhages and exudates may be found.

Basilar meningitis of a chronic type due to syphilis frequently causes secondary optic neuritis, some extra-ocular muscle paralysis, usually of the lateral rectus, and diplopia; one or both pupils may be dilated and fixed to light, with or without disturbances of accommodation and cerebral symptoms. The diplopia often induces the patient to seek advice. Optic atrophy may be the first recognized sign of a former syphilitic neuritis.

The optic neuritis due to nephritis or to diabetes is accompanied, and sometimes overshadowed, by hemorrhages and exudates in the retina. Additional causes exist, but they leave no particularly distinguishing features in the disk. The *treatment* of optic neuritis is governed by the cause.

Pseudoneuritis (see Plate X, Fig. C) often resembles optic neuritis and papilledema. There is some elevation, but no edema, no hemorrhage, and no change in the retinal arteries. The retinal veins are often congenitally large and tortuous. Pseudoneuritis is observed in some hyperopic eyes. Ophthalmoscopically it is not differentiated by the direct method, but by the indirect. The disk margins are blurred by the former, but are fairly definite by the latter method.

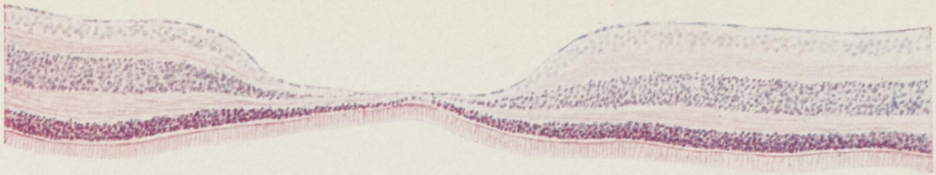
The nasal margin of the disk may be blurred without pathological significance; this is observed in many normal eyes.

Papilledema or choked disk (see Plate X, Fig. B) is distinguished from optic neuritis by several features. The disk is elevated above the plane of the retina by at least 2 diopters; hemorrhages with or without exudates are usually present about the disk; the surface is marked by radial striations from edema; the arteries are contracted and the veins are enlarged, and both wind in and out of the swollen tissues. The veins are generally tortuous over the retina. Many previously invisible veins become visible. The congestion subsides and atrophy begins. Papilledema persists for weeks or months, and may be prolonged up to two years. It may recur.

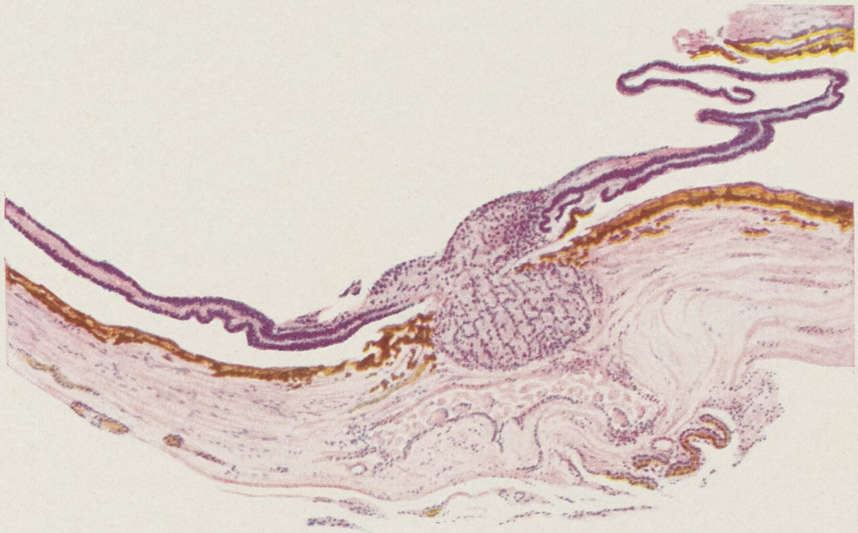
Central vision is unimpaired for a time, but concentric contractions and sector scotomata restrict the fields. Mariotte's blind-spot is enlarged. Blindness usually follows the atrophic stage.

A papilledema, followed from its beginning, presents an orderly sequence of changes due to progressive edema. Injection of the capillaries heightens the color; blurring of the borders above and below extends to the nasal margin

A



B



C

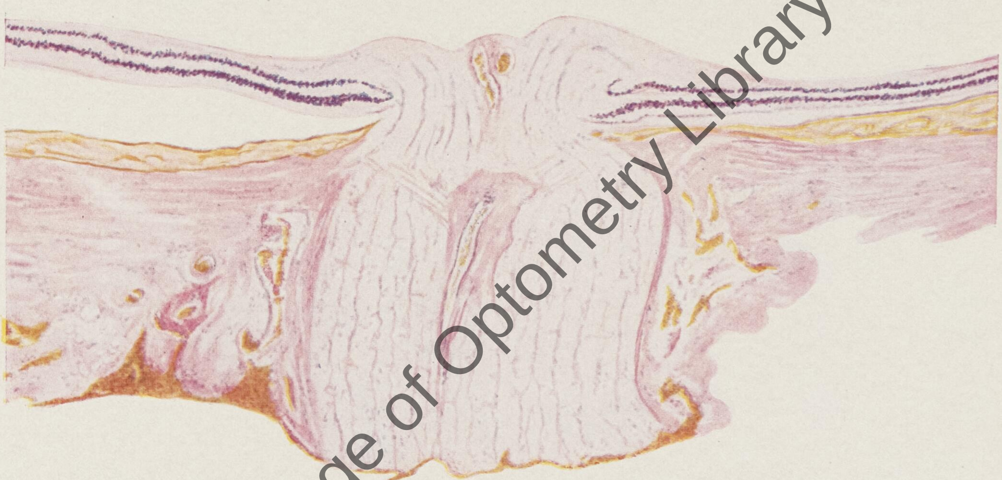


Plate X.—Fovea; Papilledema; Pseudoneuritis.

A, Fovea from monkey eye (Badertscher's specimen).

B, Papilledema from brain tumor. The tissues of the disk are infiltrated and the retina is displaced from the disk; the blind-spot of Mariotte was enlarged.

C, Pseudoneuritis. The tissues are normal and the blind-spot was not enlarged.

and finally to the temporal margin; the physiological cup fills up; the larger veins become engorged and blood extravasates from them; the elevated disk becomes grayish or opaque; exudates form about it and in the retina. Finally, the vascularity subsides, the disk becomes pale, degeneration occurs in spots, and the blood stream is contracted from thickening of the arterial walls.

A papilledema has the form of a mushroom head set over the disk. In albuminuric retinitis the picture is very similar, but the star figure about the macula in papilledema is usually limited to radial lines which point from the fovea toward the disk.

Papilledema has been found in a variety of conditions such as intracranial tumors, tumors of the orbit and intra-orbital tumors of the optic nerve, sinusitis, nephritis and internal hydrocephalus.

Papilledema accompanies 80 to 95 per cent of intracranial tumors according to statistics. Various theories have been formulated to account for its production by intracranial pressure; none has met with universal acceptance, but it is probable that each one is a factor of importance in some particular type of cases. Pressure alone is insufficient; Gowers stated that chronic hydrocephalus causes the highest intercranial pressure known, and that papilledema with it is rare and never intense.

Cerebellar or subtentorial tumors, abscesses and edemas, or hemorrhages in the posterior fossa have an earlier incidence of papilledema than occurs from similar conditions elsewhere in the cranial cavity. Tumors of the quadrigeminal bodies or midbrain are practically always accompanied by it. These facts are significant. The cerebellar fossa, which is formed of bone and covered by rigid tentorium, is open only toward the brain stem and the posterior extremity of the fourth ventricle. Sufficient pressure at this point can prevent drainage from the ventricular system into the cisternæ. Quadrigeminal tumors can occlude the cerebral aqueduct with a similar effect.

These occlusions of drainage produce an *internal hydrocephalus*. This affects the third ventricle more than the laterals because of its thin yielding floor, its extension into the infundibulum and its proximity to the openings between the cranial cavity and the orbit. An internal hydrocephalus increases the dimensions of the cerebrum and diminishes the capacity of the cisternæ. Drainage of the cerebrospinal fluid or lymph is retarded or obstructed.

The fluid is forced into the sheaths of the optic nerve and distends them. The central vessels of the retina cross the space between the sheath and the nerve. The arterial pressure is sufficient to maintain the lumen of the artery. The pressure in the vein is insufficient to maintain its lumen until it is distended by accumulated blood above the site of the obstruction or pressure, which is within the intervaginal space in this case (Fig. 151). The engorgement of

the vein favors the transudation of fluid through its walls, and this is prone to occur in the tissues of the disk.

Another source of venous stasis from back pressure occurs in thrombosis of the cavernous sinus or even of the lateral sinus. The central vein of the retina drains into the cavernous sinus directly or by way of the ophthalmic. In cases of thrombosis of the sinus the central vein becomes distended as it does when the optic nerve sheath is filled with fluid under pressure. Collateral drainage by way of the angular vein or the pterygoid plexus is often insufficient to relieve the congestion in the nerve head.

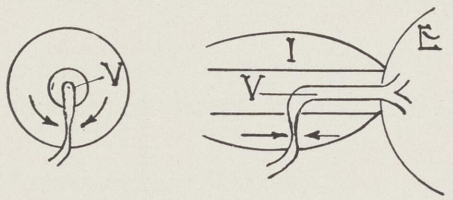


Fig. 151.—*E*, Eye; *I*, Intervaginal Space; *V*, Central Vein of Retina.

The free communications between intracranial sinuses suggest the improbability of papilledema from thrombosis of a lateral sinus, yet such is possible. Either lateral sinus may afford the principal venous drainage from the cranial cavity, while its fellow of the opposite side is small enough to be classed as an accessory. Communications between the sinuses of the two sides of the head may be inadequate to prevent congestion on the affected side. In either case there is a possibility of bilateral papilledema.

The pressure and venous stasis theories are strengthened by the behavior of the disks in the presence of tumors of the hypophysis or of the frontal lobes of the cerebrum. When such tumors exert pressure against the intracranial end of one optic canal, the cerebrospinal fluid cannot enter the intervaginal space in excess and no papilledema can occur; but pressure atrophy of the nerve may be expected on that side. Smell may be disturbed. The pressure is also exerted against the ophthalmic artery in the optic canal, and the retinal arteries may be attenuated on that side; papilledema can occur on the unobstructed opposite side.

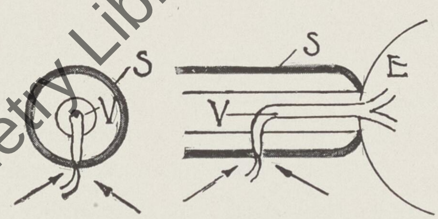


Fig. 152.—*E*, Eye; *S*, Sheath of Optic Nerve; *V*, Central Vein of Retina.

Meningitis may extend to the sheaths of the nerve and produce papilledema. The membranes are inflamed and lose flexibility. The soft central retinal vein passes out through a slit in the sheaths. The rigid inflamed wall tends to flatten or pinch the vein at this point and obstruct it (Fig. 152). The effect is the same as in the case of a cavernous sinus thrombosis; fluids infiltrate the nerve head.

Papilledema attracts attention to possible intracranial disease. Intracranial pressure is usually accompanied by headache, vomiting which is often projectile

in type with or without nausea, alteration of pulse rate, and other symptoms of irregular frequency and variable intensity.

Papilledema may appear on one or both sides, but it is seldom bilateral to an equal degree except with lesions situated in the posterior fossa or about the midbrain. One side precedes the other, and the first may be undergoing atrophy while the second is forming. Parker's experiments have demonstrated that papilledema tends to appear first in the eye which has the lower intra-ocular tension. Where it remains unilateral the lesion may be located in the orbit. As a localizing sign papilledema has only a relative value in most instances.

Differential Diagnosis *

Feature	Pseudoneuritis	Optic Neuritis	Papilledema
Elevation	Moderate	Scant or absent	Marked
Edema about disk	Absent	Present	Present
Vessels:			
Arteries	Normal	Normal	Small
Veins	Broad, tortuous	Broad, tortuous	Engorged
Hemorrhages	Absent	Rare	Frequent, usual
Vision	Hyperopic	Impaired early	Impaired late
Visual field	Normal	Central scotoma	Variable, changeable

* In each condition the color is too red, and the disk margins are obscured. Table modified from Adam, *Ophthalmoscopic Diagnosis*.

Lead Poisoning, when especially severe, may be accompanied by symptoms which resemble those of brain tumor. Wrist-drop and the blue line on the gums should be looked for. The red corpuscles show striations.

While giving a course of *tryparsamid,* the disk should be observed and the blind-spot should be plotted regularly. These should be done before beginning treatment and before giving the third dose. Optic neuritis occasionally appears after the second dose is given. This can be due to slow elimination and consequent accumulation of arsenic; small doses are indicated until the congestion of the nerve subsides. Duane, in speaking of salvarsan, ascribed the neuritis to the liberation of spirochætæ or their toxins rather than to the drug; he advised crowding the salvarsan and the use of mercury concurrently.

A descending neuritis is illustrated by the changes that occur in the disk in association with a meningitis; the disease reaches the eye by way of the optic nerve. Not every case is ophthalmoscopically demonstrable.

The Trunk

Retrobulbar neuritis and axial neuritis are varieties of inflammation which affect the nerve trunk without characteristic eye-ground changes; the fundus may be normal in appearance. Retrobulbar neuritis is acute or chronic.

The chronic form is referred to as toxic amblyopia or as axial neuritis. The term "retrobulbar neuritis" ordinarily refers to the acute type.

Retrobulbar neuritis is induced by general and local causes; disseminated sclerosis, meningitis, cerebral abscess, focal infections, exposure to cold and wet, the infectious diseases, lead poisoning, syphilis and diabetes may cause it. Infections or inflammations of the orbit or accessory nasal sinuses are frequently responsible. It has been caused by hemorrhages into the sheath of the optic nerve, and by thromboses of orbital veins.

Acute retrobulbar neuritis is mostly unilateral; it is generally accompanied by some headache. The globe may be proptosed from swelling of the nerve or from congestion of the orbital contents; attempts to press the globe back are attended by pain. The tendons of the superior and medial recti are adherent to the sheath of the optic nerve at the posterior part of the orbit normally, and attempts to rotate the eye often cause pain. The eye-ground is usually negative in the early stages although an optic neuritis may come on late. The retinal veins are engorged.

The most reliable evidence of retrobulbar neuritis is afforded by failing sight. The failure is usually rapid; it often goes on to complete blindness, and the blindness may be permanent. The peripheral field is usually contracted. Sometimes only the macular and paramacular areas are affected, with resulting central and paracentral scotomata. Perception for red and green is lost before that for blue and white. Arrest of the disease is ordinarily attended with restoration of some vision, although defects are prone to persist.

Similar defects of vision are found in disseminated sclerosis, but they come on more slowly. In the latter, the form field is usually full, and the central scotoma is relative or for colors only. In retrobulbar neuritis there are generally some contractions of the form field, and the central scotoma is absolute or for both colors and white. The fields should be tested with small differently colored objects. True nystagmus and disturbances of reflexes point to disseminated sclerosis. Hysteria should be excluded.

The behavior of the pupil has significance. Normally the pupil contracts to light, then dilates, contracts again and finally dilates to some extent a second time. In retrobulbar neuritis the secondary contraction is seldom present.

The treatment depends upon the cause if known; otherwise mercury, iodids, diaphoretics and other eliminants are indicated. When focal infection is suspected, or much pain is present, salicylates or acetyl salicylic acid are indicated. The prognosis should be guarded. Recovery of some useful vision is the rule, but it cannot be promised. Optic atrophy is a not uncommon termination. The disk will sometimes show postneuritic atrophy without the neuritis having been recognized.

Toxic Amblyopia and Axial Neuritis are designations for chronic retrobulbar neuritis. Axial neuritis is descriptive since the fibers which supply the macular region are affected. These fibers enter the nerve on the temporal side of the disk (see Fig. 150). After leaving the eye they occupy the center or axis of the nerve.

The earliest subjective complaint is misty vision or a dazzling which is worse in bright light and less noticeable on cloudy days or in the dusk. The patient gradually loses the ability to read fine print or to do fine work because central vision is defective. The resulting scotoma is negative or unknown to the patient.

The objective evidence is demonstrated by a central scotoma. The fovea is represented at the point of fixation. In axial neuritis the scotoma consists of an enlargement of the blind-spot in the direction of the fixation point, and in some cases that point is covered by the scotoma. A blind area that covers both blind-spot and point of fixation is pathognomonic; it is usually horizontally oval. The affection is nearly always bilateral.

Central blindness or a central scotoma for red and green is confirmed by a direct visual test. A simple contrivance consists of a cylinder about 4 centimeters ($1\frac{1}{2}$ inches) in diameter and 10 to 12 centimeters (4 or 5 inches) long; it is lined with photographers' paper and the ends are closed. The black trade cartons for 1 ounce round bottles of pharmaceuticals do nicely. A pinhole is made in the center of each end. The patient closes one eye, and with the other looks through the two holes at a light or the sky. When the light is perceived, red and green glass are held alternately in front of the cylinder and the patient is told to name the colors.

This test is not strictly accurate when negative, since the patient may be sighting with an extrafoveal region of the macula. Before making this test the colored lenses should be held between the patient's eye and the light to confirm the presence of color perception. The test is valueless where colors cannot be recognized.

The same test can be used for the detection of central chorioretinitis, but with a blue glass. Distortion of objects from edema of the choroid or retina will be present; this does not occur in axial neuritis. In macular disease the scotoma is positive; the patient is conscious of it in his field of vision. In axial neuritis the scotoma is negative.

In early stages the ophthalmoscope usually reveals a hyperemic disk. Later, the temporal quadrant of the disk becomes unusually white or pearly, and rarely the entire disk may look like secondary or white atrophy. Special forms of poisoning may show somewhat characteristic appearances.

Pathologically, there is an interstitial neuritis of the papillomacular bundle

which is followed by atrophy of the nerve elements and a proliferation of connective tissue. The changes are analogous to those that occur in the brain in cases of chronic alcoholism (Swanzy).

Causes of the disease are many. In the foregoing discussion tobacco and alcohol poisoning have been more particularly in mind. Either substance can cause it, but the two in combination are more effective. The susceptibility of the patient is a greater factor than the amount of the agent used. Other drugs which have been responsible are carbon disulphid, dinitrobenzol, atoxyl (see tryparsamid), felix mas, cannabis indica, stramonium, opium, salicylic acid, quinin, arsenic, lead, chloral, iodoform and aniline dyes. Several of these may be classed with occupational diseases.

Treatment consists first in the removal of the offending substance by correcting injurious habits or changing occupation. General treatment includes nitroglycerin, strychnin, and potassium iodid in large doses. Pilocarpin sweats, Turkish baths, saline catharsis and the drinking of large amounts of water are indicated. Treatment must be persistent and continuous. The *prognosis* depends upon the amount of damage sustained. Restoration of vision may be almost complete or the disease may progress to permanent blindness.

Diabetes is sometimes complicated by axial neuritis. The prognosis here depends upon the severity of the disease. Axial neuritis has been observed in syphilis, in some diseases of the nasal accessory sinuses, in some febrile diseases, and after deep burns of large areas of the skin. In these an actual neuritis may sometimes be demonstrated ophthalmoscopically.

Drinking wood alcohol or inhaling its fumes produces symptoms of retrobulbar neuritis which often affects the entire nerve. Visual depreciation sometimes begins within twenty-four hours. The central scotoma is absolute and often positive. The peripheral fields are contracted. Absolute blindness may rapidly follow. The pupils are dilated and fixed to light. The eyeballs are tender to pressure and attempted rotations cause pain. Visible optic neuritis is succeeded by optic atrophy. Constitutional symptoms are present according to the degree of poisoning. Vision is often partially restored temporarily, but permanent blindness may be expected. However, some cases do recover both useful vision and health.

Quinin poisoning is discussed under diseases of the retina.

OPTIC ATROPHY

The term optic atrophy generally refers to an atrophy that affects the disk and is demonstrable by the ophthalmoscope. It is a degeneration, usually bilateral, and is marked by reduction of vision. It is classified according to

etiology into primary, secondary and consecutive types. These cannot be assuredly differentiated by the ophthalmoscope, because changes occur whereby each type may come to resemble the others.

Primary, simple, or noninflammatory atrophy occurs without antecedent primary disease of the nerve itself so far as can be demonstrated. The secondary or inflammatory type follows optic neuritis and papilledema. The consecutive type follows some disease that is primary in the retina or choroid.

The color, margins, and level of the disk, and the disposition and character of the blood-vessels are significant in atrophies. The ophthalmoscope light should be neither too bright nor turned full upon the disk, but to one side first in order to avoid reflections and to gain the advantage of contrast in light and shade. The visual functions should be investigated in all cases.

Primary Atrophy.—The disk color is generally gray, as the stippling of the lamina cribrosa is visible. The area of the stippling depends upon how much of the lamina can be seen. The disk may be tinted with green or blue. The color often changes in time from gray to white, or from white to gray. The disk margins are sharp, and the contrast between the atrophic disk and the retina, which may appear normal, is marked.

The disk is concave like a shallow saucer, but this is difficult to detect with the ophthalmoscope. The pallor of the disk is due to the disappearance of its capillaries. The retinal vessels are unchanged, or the arteries may be small.

Secondary Atrophy.—The disk is generally a dense paper white. This is due to proliferation of connective tissue in the organization of an inflammatory exudate. This tissue conceals the lamina cribrosa, extends over the margin of the disk, disturbs the peripapillary pigment and invades the perivascular sheaths of the retinal vessels. The margin of the disk is indistinct and irregular in outline.

The disk capillaries disappear or are concealed. The retinal arteries are small, while the veins may be normal or small and wavy. Contraction or further proliferation of connective tissue in the perivascular sheaths will narrow the blood column in time. This tissue shows as white lines along both sides of the vessels. The small retinal vessels may disappear. The contraction of the connective tissue often gives the nerve head a shrunken appearance. In such cases it is surrounded by a ring of sclera. Where the connective tissue is thin the disk will look grayish, greenish or bluish; it may or may not be cupped.

Consecutive Atrophy.—Where retinitis or choroiditis is the cause the exudate and capillaries give the disk an early appearance of mixed gray and red. The color changes to yellowish or waxy, but in time it will resemble that of other atrophies (see Plate VII). The margins become indistinct from earlier

changes that had occurred in the retina or choroid next the disk. The few visible retinal vessels are narrow. This is especially true in blind areas of the retina and in retinal degeneration.

In all cases of all types the vision is affected. In the primary type the prognosis is bad. In the secondary and consecutive types the prognosis depends upon the amount of damage done before the process is arrested. A considerable restoration of vision is possible. Concentric or irregular contraction of the peripheral fields occurs early. The contraction of the green and red fields is more rapid than that of the form field. Color-blindness and form blindness follow the disappearance of the respective fields. Scotomata usually precede the stage of blindness. Central scotomata alone characterize some cases of atrophy.

Optic atrophy has been traced to a variety of causes. The primary type occurs from a loss of continuity in or a loss of nerve-fibers. The nerve may be severed in basal fractures of the skull; total blindness is immediate, but the atrophy is delayed.

Compression on the intracranial portion of the nerve or chiasm by tumors, a distended third ventricle in internal hydrocephalus, an atheromatous internal carotid artery or an aneurysm of that artery or compression on the canalicular portion by an atheromatous ophthalmic artery may cause atrophy, or it may be due to hemorrhage into the sheath of the nerve, gross hemorrhage in the orbit, orbital cellulitis, nasal accessory sinus disease, pituitary disease, meningitis or glaucoma. Some of these causes may seem to be productive of the secondary type, but all of them interfere with or interrupt conduction. Inflammatory products complicate the situation; these and pressure can combine to produce a mixed form of atrophy.

Pressure on or disease of any part of the nerve, chiasm or tract from the globe to the external geniculate body may be responsible for an optic atrophy. In the conditions enumerated but one eye may be affected. The disease, when due to constitutional causes or to general pressure on the chiasm, is bilateral in practically all cases.

Pressure on or destruction of one optic nerve produces an atrophy of that disk only; where the lesion divides the chiasm anteroposteriorly the nasal halves of both disks atrophy. When the lateral sides of the chiasm are damaged, as by bilateral aneurysms of the internal carotids, the temporal halves of both disks atrophy. Where the lesion affects but one optic tract the atrophy will appear in the corresponding halves of both disks; the lesion and the atrophy will be either on the right or the left side. For field defects corresponding to these lesions, see hemianopsia.

The optic atrophy of locomotor ataxia is the classical example of the

primary form. Then follow general paralysis of the insane, disseminated sclerosis, syphilis of the central nervous system, various disorders of the cerebrospinal system, arteriosclerosis, renal disease, diabetes, acromegaly, oxycephaly, malaria, embolism of the central retinal artery, thrombosis of the central retinal vein, and the causes of axial neuritis as tobacco, alcohol, quinin or wood alcohol; a number of reported cases have occurred without discoverable etiology.

The pathological changes in the primary form consist of the atrophy and loss of nerve-fibers. In the secondary form there is also an atrophy and loss of nerve elements, but in the organization of the inflammatory materials, connective and glial or interstitial tissues are proliferated. These fill the physiological cup, conceal the lamina cribrosa, veil the margins of the disk, and follow the sheaths of the retinal vessels.

Tabetic Atrophy of the optic nerve is generally the earliest sign, sometimes by years, of tabes dorsalis, locomotor ataxia or posterior sclerosis. When this atrophy occurs very early, ataxia may not develop at all, or only very late. The optic atrophy itself does not protect from ataxia (Parsons), but when it occurs late in the disease, ataxia will develop in the usual course of events. When blindness comes on within two or three years it seems to retard the development of ataxic symptoms, as they are seldom found in tabetic blindness.

Locomotor ataxia is characterized by a history of syphilis, Argyll Robertson pupils, loss of patellar reflexes, visual defects, paralysis of extra-ocular muscles, cutaneous anesthesia, especially in the distribution of the trigeminus, and incoordination or ataxia.

In early stages there is spinal miosis, or contraction of one or both pupils from paralysis of the cervical sympathetic nerves. The form of the pupil may be irregular. In later stages there is less sensitiveness to small differences in illumination on account of degeneration of the optic nerve and retina. As light sensitiveness diminishes or conductivity decreases the pupil dilates and becomes immobile to light. The pupillary phenomena are bilateral in practically all cases, although the pupils are unequal in size and reactions where one eye is affected before the other.

The pupillary contraction associated with convergence is independent of the optic nerve and retina, and so the convergence reaction is preserved unless there is a lesion of the motor oculi or its nucleus. Consequently, the pupils may contract with convergence when the atrophy is complete and the eyes are blind. Where but one optic nerve ceases to function, the pupil on that side will not react to direct light, but the consensual or indirect light reflex will be present and may be exaggerated.

The site of the lesion for the Argyll Robertson pupil is thought to be in

the tissues around the cerebral aqueduct or in the floor of the third ventricle. The atrophy is caused by an inflammatory exudation into the intracranial portion of the optic nerve and the chiasm (Parsons). There are a gray degeneration and a proliferation of glial tissue which produce changes similar to those that occur in the posterior columns. The part of the nerve attacked and the extent of the lesion will determine the type of visual defect. The disk shows some atrophy before the vision is appreciably diminished.

The visual deficiencies associated with tabetic atrophy are manifold. There is always a diminished ability to detect small differences in illumination. Central vision deteriorates unequally in the two eyes; the refraction should be closely corrected for making comparative tests. One or both form fields are contracted concentrically, irregularly, with reëntrant angles, with quadrant anopsia, or with hemianopsia. Central or paracentral scotomata may be found. The color fields contract more rapidly than the form fields, and color-blindness comes on long before form blindness. Green, red, blue and yellow perception are lost in the order named. Deterioration of vision is marked by remissions and exacerbations.

Paralysis of the lateral rectus associated with primary atrophy of the optic nerve is suggestive of tabes.

General Paralysis of the Insane is considered by many to be of syphilitic origin. Primary optic atrophy may precede other evidence of the disease as it does in locomotor ataxia. Pupillary changes are early and significant. There is marked inequality of the pupils which are often irregular in shape. The light reflex is absent, and there is often a loss of the convergence reflex.

Cerebral Syphilis is believed to be associated with syphilitic basal meningitis, possibly gummatous, with involvement of the intracranial portion of the optic nerve, chiasm or optic tract. Optic neuritis or papilledema is followed by atrophy of the nerve and disk. Bilateral paralysis of the motor oculi nerves with ptosis is not uncommon. The trigeminus, abducens and facial nerves may be affected. Vision is seriously reduced. Homonymous hemianopsia (see Fig. 124) often occurs. Central scotomata and total atrophy of the disks are found occasionally. The symptoms vary both in character and intensity from time to time. The disease also attacks the cerebral blood-vessels.

The type of atrophy found in locomotor ataxia, general paralysis and syphilis of the nervous system does not serve to differentiate them. The pupils are helpful. In locomotor ataxia the pupils are small and immobile to light but react to convergence. The eyes are not affected to the same degree. When blindness is established the pupils will dilate, equally as a rule, and the convergence reflex will be lost.

In general paralysis the early signs will be similar to those found in tabes,

but there will probably be more difference in the size of the pupils, which are often misshapen. Dilatation is established early and the convergence reflex is lost much sooner than in tabes.

In syphilis of the nervous system usually but one eye is affected. That pupil reacts neither to light nor to convergence, and paralysis of accommodation is an ordinary complication.

Traquair (*Clinical Perimetry*) states that in tabetic atrophy the pallor of the disk progresses along with visual failure, while in syphilitic perineuritis the failure of vision precedes the appearance of pallor.

Disseminated Sclerosis is accompanied by a partial atrophy of the primary type. The axis cylinders are preserved but their sheaths are attacked. This leads to variations in the visual defects from time to time, for only the conductivity of the nerve is disturbed, and functional restoration is possible. Blindness is rare. While the fields may vary, there is little or no permanent loss in the periphery. Central scotomata are relative and negative; the patient is unaware of them. Visual failure is usually unilateral. Nystagmus and paralysis of convergence with retained ability to perform lateral rotations are suggestive of this disease.

In disseminated sclerosis vision is affected before the atrophy appears in the disk; the reverse is true in tabes.

Arteriosclerosis, atheroma, senile pallor of the disk and nutritional atrophy of the optic nerve are allied. The vision is ordinarily good. There is no cupping. Faint markings of the choroid surrounding the nerve head indicate changes in the choroidal vessels in that locality. The retinal vessels are often irregular in width. In the atrophy following neuritis and papilledema the disk is white, its margins are obscure, it may be slightly elevated, and it often appears to be shrunken in diameter. The veins are usually large and tortuous and the arteries are small. Connective tissue is found in the vessel walls and in the region of the disk.

Atrophy follows embolism of the central artery. The vessels are few and thready, and the eye is blind from the onset of the embolism. Atrophy following thrombosis of the central vein is accompanied by permanent changes due to hemorrhages and local atrophies in the retina.

In retinitis pigmentosa, with or without pigment, there is a consecutive atrophy of the disk. The margins are indistinct, the color is waxy or yellowish, and the retinal vessels are few and thready. A yellowish disk is sometimes seen in congenital night-blindness.

In partial atrophies such as follow toxic amblyopia or axial neuritis, the pallor is largely limited to the temporal quadrant of the disk. A central scotoma without peripheral contraction of the fields is present. It is a bilateral

affection. Unilateral partial atrophies occur in multiple sclerosis and in some diseases of the accessory sinuses of the nose.

Glaucoma is accompanied by atrophy. This is gray or dirty white in color. The lamina cribrosa is not covered, but is pushed back into the nerve sheath (see Plate XI). The margins of the cup thus formed are sharp or abrupt as shown by the vessels as they bend over the edges. Often the glaucomatous halo, which is not to be confused with the halo surrounding artificial lights, encircles the disk. It is due to a thinning of the retina from traction in that vicinity. In late stages a proliferation of connective tissue may change the color of the disk from gray to white.

In glaucoma the form and color fields contract proportionately; this distinguishes the process from that of other forms of atrophy in which the color fields contract more rapidly than the form fields. In glaucoma the sensitivity for light is diminished; more than the ordinary amount of light is required to stimulate the retina. In optic atrophy the retina is sensitive, but small changes in the intensity of light are not detected as readily as they are in glaucoma.

In cavernous atrophy of the optic nerve with partial excavation of the disk, the color fields disappear more rapidly than the form field as they do in other optic atrophies. The caverns, lacunæ or Schnabel's spaces are also found in some cases of glaucoma and of high myopia.

Differential Aspects of Various Atrophies as Seen by the Ophthalmoscope *

Type of Atrophy	Color	Margins	Depression	Vessels
1. Nerve severed	White, uniform	Sharp	None	Normal
2. Retinal anemia †	Pallid	Distinct	None	Normal
3. Retinitis pigmentosa	Yellowish or waxy, reddish gray	Obscured	None	Very fine
4. After embolism of central artery	White	Distinct	None	Very fine
5. Simple or tabetic	Stippled bluish gray	Sharp	None or saucer	Normal, or arteries small
6. Postneuritic	Uniform, white	Blurred irregularly	None; filled in	Small; white borders
7. Postpapillitic	Dirty white	Indistinct	None or elevated	Small; white borders; veins tortuous
8. Postaxial neuritic	Grayish or white on temporal side	Distinct	None	Normal
9. Postglaucomatous	Grayish	Distinct, with halo sometimes	Abrupt at margins	Hook over margins; veins engorged, tortuous; arterial pulse

* Modified from Adam, *Ophthalmoscopic Diagnosis*.
† Retinal anemia is not an atrophy, but may be readily mistaken for it.

Hereditary Optic Atrophy or Leber's disease usually appears in males between the ages of thirteen and twenty-eight years. It is transmitted by females who are generally but not entirely exempt. It affects the papillomacular bundle principally, although it may involve the entire nerve. Pallor may appear in the temporal quadrant or include the whole nerve head. It is a bilateral disease, but it may affect one eye earlier than the other.

Failure of vision comes on rapidly and sometimes abruptly. The process is gradually retarded and may become stationary; after that a progressive improvement leads to partial recovery. On the contrary, the tendency may take the opposite direction toward blindness. Total permanent color-blindness is not uncommon. When several members of a family have the disease the characteristics are similar in each.

The disease is essentially a retrobulbar or an axial neuritis. In early stages edema and congestion may appear in the disk and blur its edges; a gray discoloration appears later, and finally there is a definite atrophy. The principal visual defect is a central scotoma, especially for colors; the field may show some concentric contraction.

The cause has not been determined. Some investigators suspect disease of the nasal sinuses. Because of the age at time of onset it has been attributed to endocrine disorders. It is occasionally associated with palpitation of the heart, vertigo and migraine. The sella turcica should be investigated by roentgenography. The treatment is unsatisfactory. Where endocrine functions are believed to be at fault, pituitrin or thyroid extract may be tried. Mercury, iodid of potassium, strychnin and galvanism have been used.

ANOMALIES AND INJURIES OF THE OPTIC NERVE

Tumors of the optic nerve or of its sheath may be primary or secondary. The globe is proptosed and immobile; pressure against it does not usually produce pain. When the tumor affects the nerve early, blindness may be expected; this is not ordinarily true of tumors of the sheath. Diplopia is not an uncommon symptom. These tumors are sometimes removed with preservation of the eyeball by the Krönlein operation (see Fig. 78) or by Knapp's operation.

In the Krönlein operation the temporal wall of the orbit is temporarily resected. In Knapp's operation the medial rectus is temporarily detached. Each method was devised to afford access to the nerve without removing the globe.

Massive tubercle of the nerve head is larger than the disk and projects into the vitreous. It is one of the pseudogliomata and must be differentiated from intra-ocular glioma and leukosarcoma. The usual tests are suggested. A course of tuberculin reduces this growth rapidly.

Coloboma of the nerve or of its sheath is rare. The disk area is materially enlarged, and the retinal vessels seem to arise from the edge of the *white* region. It is usually continuous from and associated with coloboma of the choroid and retina.

Injuries of the nerve may be due to direct violence, as by wounds that enter the orbit, or by falls or blows that cause concussion or that produce basal fractures of the skull. When the concussion occurs direct to the eyeball it usually results in rupture of the choroid with or without evulsion of the nerve. Evulsion of the nerve means a partial or complete tearing of the nerve from the eyeball. A partial evulsion may completely separate a part of the circumference of the nerve, or it may show as a partial separation of the whole circumference of the disk (see Plate VIII, Fig. E). Intracranial hemorrhage may enter the sheath of the nerve and produce blindness.

CHAPTER XIII

THE VITREOUS

DESCRIPTION AND DISEASES

The vitreous body is a transparent, colorless, jellylike substance which occupies all of the space in the eyeball behind the lens. It holds the retina in contact with the choroid. The semifluid material is supported by a meshwork of delicate fibrils that have no nuclei. The meshwork is condensed in the anterior part where it is adherent to the pars plana of the ciliary body and the posterior capsule of the lens. This portion is called the anterior border layer. The lens lies in the patellar fossa in the center of this layer. The vitreous body is said to be enclosed by the hyaloid membrane.

The vitreous is devoid of blood-vessels and nerves; it is nourished probably by the aqueous humor. There are no truly organized elements in the vitreous, except wandering cells and occasional leukocytes.

In fetal life the hyaloid artery extends from the disk to the posterior surface of the lens. After birth the sheath of this vessel sometimes persists as the hyaloid canal. The sheath is always attached to the disk; the anterior end either floats free or is attached to the posterior surface of the lens. It may serve as a lymph channel. It can be traced from the disk forward ophthalmoscopically by using convex lenses of gradually increasing strengths. A demonstrable sheath is seldom present.

Lacking blood-vessels, hyalitis or true inflammation seems impossible. Yet an irritant may cause symptoms of inflammation. Otherwise disease of the vitreous is secondary. Fluidity, opacities, foreign bodies, hemorrhages and abscesses are the principal pathological entities found in the vitreous.

Opacities of the vitreous are of various kinds; they may be apparent to the patient but undemonstrable to the examiner, or they may be the reverse. They may be small or large, round or elongated, and few or abundant. They are generally free or floating and are rarely fixed. To search for them the pupil should be dilated and the room darkened. The examiner stands 12 to 18 centimeters (5 to 7 inches) in front of the eye and directs the light of the ophthalmoscope toward the disk, which makes a suitable background for demonstrating floaters. Convex lenses of 4 to 7 diopters are used in the sight hole. Fine

bodies are best seen with low illumination; they are often invisible in bright light.

The commonest form of opacities is muscæ volitantes. They are projected into space as entoptic images. Semitransparent elements in the vitreous, or corpuscles in the retinal vessels may produce them. They can be demonstrated usually by looking intently at a bright surface, at a bright light, or at a brightly illuminated page of print, and are often very annoying when using a microscope.

They appear as pale gray shadows of globules, pearls, or rings, grouped or in chains, and they follow the movements of the eyeball, especially in reading. They rarely appear directly in the line of vision, but a little to one side. They may also be seen by looking through a narrow slit at a white cloud. They are most readily seen by suddenly moving the eyeball, and then looking blankly toward a bright reflecting surface. Muscæ float about in fantastic patterns. Myopic eyes detect them easily, for the images are larger in this affection. They cannot be demonstrated ophthalmoscopically.

Muscæ are not pathological. The patient should be advised to ignore them, for they are usually forgotten after a time. When they persist, the patient is advised to rest from near work. Ametropia should be corrected. Gastro-intestinal disorders should have attention.

Demonstrable opacities of the vitreous exist as very fine dust, as flaky or thready masses, or as bands from the retina into the vitreous. The dust is regarded as good evidence of antecedent syphilitic disease. Flakes and threads follow the organization of exudates and hemorrhages. Bands are formed from connective tissue proliferation in the healing of wounds of the retina or choroid, and are usually classed as proliferating retinitis.

Opacities result from high myopia, injuries to the eye, diseases of the retina or uveal tract, systemic diseases, senility, and without known cause. They disturb vision little or much according to their number and size. When they are large and float into the line of direct vision, the patient must dislodge them by a movement of the eye before seeing clearly again. In taking the manifest vision it may be noted that the patient varies materially in ability to read the types. With each movement of the eye the opacities are put in motion. Frequently the patient knows this.

Ophthalmoscopically, the fundus will seem redder than normal in the presence of fine opacities; the disk will appear especially red, like the sun in a light fog. Opacities often obscure the fundus; the examiner must be guarded in interpreting details in this circumstance.

Treatment depends upon the cause; mercury or iodids may help to clear up the opacities. Subconjunctival injections of normal salt solution are useful

when there is no active inflammation. Dionin is instilled in 1 to 5 per cent solution, one drop in the eye at night. The general health must have attention. The prognosis depends upon the cause.

Asteroid Hyalitis consists of a vitreous filled with fixed and movable dull white spheroid bodies that resemble snowballs. They vary in size; they reflect light but do not glitter. They occur in one or both eyes of elderly persons and are persistent. The cause is unknown; they have not seemed to cause damage, and no treatment has been devised to remove them.

Synchysis, or fluidity of the vitreous, is a condition of hypotony or soft eye which is occasionally found in the aged. Opacities must be present to make the diagnosis positive. Crystals of cholesterin are seen as a shower of glittering gold or silver particles when the eye is moved. This is called *synchysis scintillans.* The intra-ocular tension is reduced. There is no known cause. It is often found in myopia. It has no bad effects, and no treatment is indicated.

The importance of fluid vitreous is apparent in the extraction of cataract. When the globe is opened the vitreous may prolapse or the lens may be dislocated before it can be delivered. A moderate loss of such vitreous is said to be of less importance than a similar loss of normal vitreous. Fluidity of the vitreous may facilitate detachment of the retina.

Hemorrhages into the vitreous come from intra-ocular blood-vessels. When they are particularly massive nothing can be seen behind the lens. The pupil is jet black. Oblique illumination sometimes shows a red reflex from the posterior surface of the lens. Small hemorrhages may be visible with the ophthalmoscope.

Hemorrhages are due to injuries, diseases of the choroid and retina, myopia, glaucoma, high blood-pressure, systemic diseases, and atheroma. Some hemorrhages are spontaneous. Small masses are usually absorbed; large ones often undergo organization with the formation of retinitis proliferans or detachment of the retina. When blood pigment is deposited, siderosis develops.

Recurrent Hemorrhages into the vitreous occurs in young persons, mostly males, between the ages of fourteen and twenty usually. The blood is defective in coagulability. Very frequently there is an associated tuberculous infection. The retinal veins may show a periphlebitis. When the clot organizes connective tissue forms; such masses may detach the retina, especially as the cause is repeated. When the hemorrhage is connected with the disk, cicatricial membranous bands are much more apt to form.

Vitreous hemorrhages are treated according to the cause. Rest in bed is indicated. Calcium salts help restore the coagulability of the blood. The blood-pressure must be reduced, if high. Constipation must not be permitted,

as all straining must be avoided. Dionin and subconjunctival injections of normal salt solution are indicated. Mercury may be given. Iodids are indicated when there is no evidence of tuberculosis.

Abscess of the vitreous occurs in two forms. The milder type consists of masses of flocculent yellowish material. In the grave type pus fills the vitreous chamber. Exogenous infections are due to penetrating injuries and to retained foreign bodies. Endogenous infections arise in consequence of purulent or metastatic choroiditis, erysipelas, scarlet fever or other pyogenic infections. Meningitis is the most frequent antecedent of abscess of the endogenous type.

True abscess is confined to the vitreous. Where it is found in children it may be mistaken for glioma of the retina; it belongs to the group of "pseudogliomata." The history of recent acute illness and low intra-ocular tension constitute the essential points of differentiation. The eyeball is inflamed intensely and a yellowish mass may be seen behind the lens. Small blood-vessels have been seen on the surface of such a mass.

When the abscess involves the retina and choroid it is called *septic endophthalmitis*. Where the iris is affected or the process has reached the anterior segment of the eye, the peripheral border of the iris will be retracted while the pupillary border will protrude into the anterior chamber. In either instance, whether confined to the vitreous or extended to the anterior segment, the globe shrinks. Where the abscess perforates the coats of the eye it is called *panophthalmitis*. The globe contracts and this condition is known as "phthisis bulbi."

Unless a case can be seen sufficiently early to arrest the antecedent disease and save some vision, enucleation or evisceration is generally indicated. When the disease is mistaken for glioma there is not much loss from removing a deformed globe, since it is useless as a visual organ.

Fibrous Tissue may form in the anterior portion of the vitreous in consequence of the membranous bands that are produced during the subsidence of plastic iridocyclitis. The lens is usually opaque and such bands cannot be seen.

Parasites are infrequently found in the vitreous or behind the retina. The diagnosis depends upon recognizing some part of the parasite or detecting its movements. The treatment consists in surgical removal.

RETAINED FOREIGN BODIES AND THEIR TREATMENT

The eyeball is frequently injured, especially in industrial centers, by foreign bodies that remain inside the globe. They may lodge in the iris, lens, ciliary body, vitreous, retina or choroid; they nearly always injure the vitreous.

When they are behind the lens they are described as being in the posterior segment of the globe.

Foreign bodies consist of pieces or chips separated from larger bodies. The substances most frequently found in the eye are iron, steel, shot, copper, stone, glass and wood. A small body enters the eye with facility; a large body may rupture the coats without entering the globe. A body that has sufficient mass and velocity can pass through the eye and wound extra-ocular tissues; such bodies are often found in the orbit.

The immediate danger from a retained foreign body is infection. The penetrating body may be sterile or it may carry infecting organisms. As it enters it carries these agents in from the conjunctival sac, or it opens a pathway for them. The probability of infection includes several factors. (1) The nature of the infecting organism and its virulence in eye tissues; (2) the capacity of the foreign body to produce irritation; and (3) the resistance of the eye tissues to the particular organism, and their toleration of the presence of the foreign body.

The several tissues of the eye behave in different ways toward infections and irritations. The vitreous humor is a good culture material. The uveal tissues react intensively to mechanical irritation. In either case a violent inflammation may be expected. This may take the form of a vitreous abscess, a septic endophthalmitis or panophthalmitis. When the primary inflammation appears in the uvea an iridocyclitis is the natural reaction, and, if the organisms are nonpyogenic, there is the added danger of sympathetic ophthalmitis. Pyogenic organisms do not produce this disease.

Tissue susceptibilities vary in different ages. The resistance of the structures of young eyes is superior to that of old eyes. This factor may account for the observation that a foreign body in one case was retained for twenty-four years before inflammatory symptoms were set up; the eye was quieted by magnet extraction of a piece of iron (F. A. Morrison). Another case had no symptoms for eighteen years. It follows that the older the patient the greater the apprehension.

Foreign bodies that enter the eye may stop in the vitreous, cling to the retina or the choroid, or rest against or be imbedded in the ciliary body. They sometimes become encysted in exudates, or connective tissue membranes may form. The latter process is more probable when there has been some hemorrhage.

The foreign material may partially dissolve by chemical action. Iron has the reputation of doing this with the production of siderosis. The iris is first stained greenish, and later a brownish red. When this change appears in the lens an irregular circle of rust spots is found in it beneath the border of a well

dilated pupil. These spots are oval in shape and have a radial arrangement; the appearance is pathognomonic (Parsons).

Copper seems to have the property of exciting inflammatory reactions even though the metal is sterile at the time it enters the eye. Glass is the least offensive. Stone chips which have rough and uneven surfaces are frequently the bearers of infecting organisms. Wood is a near second; it produces a chronic inflammation with a dense granulation tissue.

When a patient presents with an injured eye the surgeon should inquire into the patient's environment and what was being done in the vicinity at the time of the accident. It is possible to learn the probable nature of the body and acquire some information about the size of it. A large wound with extruding vitreous or prolapsed iris needs no exact diagnosis, as the eye should be removed. Refinement in diagnosis is due when there are no signs of extensive injury; a foreign body may be in the eye.

The wound of entrance must be in the cornea or sclera. Small wounds may heal on the surface and be undemonstrable after twenty-four hours. When the case is seen early, the wound will stain with fluorescein. Where the wound is in the cornea, a hole in the iris or a wound in the lens can probably be located unless the anterior chamber contains blood.

A hole in the iris is black to oblique illumination, while with the ophthalmoscope the red fundus reflex can usually be seen through it if the lens is clear. A wound in the lens will likely include both its anterior and posterior surfaces.

When the lens is clear, as it will be in the first twenty-four hours, and there is no blood or other opaque material in the vitreous, the pupil is dilated and the ophthalmoscope is utilized for continuing the examination. Where the foreign body has traversed the vitreous, the path is often marked by a gray line or a row of air bubbles. It is sometimes possible to see the foreign substance.

Where the foreign body has lodged within the ciliary body, there will probably be a point of decided tenderness over the wounded tissues, more especially over the situation of the body. Tenderness may be found, however, when the ciliary body has not been injured.

A foreign body in the vitreous, retina or choroid is rather quickly concealed by a white exudate or a red blood-clot. When an intruding substance is situated in any of these parts, it may be possible to chart the exact location by a scotoma in the field of vision. White test objects are used for this purpose.

The foreign body may be magnetizable. For diagnostic purposes a hand magnet may be used. It must first be ascertained that the contact of the magnet does not cause pain; if it does, the magnet must not be allowed to quite touch the eye when the current is turned on. If iron is present it will be at-

tracted toward the magnet and cause pain. When the hand magnet fails, the giant magnet may be tried. Failure with the giant instrument constitutes presumptive evidence against the presence of magnetizable metal.

Certain changes may have occurred before the patient is seen by the surgeon. After injury, the lens is clear for a few hours; it then becomes cataractous and nothing can be seen behind it. Absorption sometimes occurs later on and the fundus becomes visible again.

The exudate or blood-clot that may encyst a foreign body usually undergoes changes due to the proliferation of connective tissue. This may merely envelop the foreign body, or it may extend as a membrane or band from it to some other part of the eye. As a rule, black pigment accumulates in the vicinity. Some pigmentary changes are occasionally found about the macula.

When the eye is filled with blood or the lens is opaque, and other methods have failed or are inapplicable, roentgenographs must be made. Exact localization by roentgenography requires a complicated apparatus. In the absence of such facilities, approximate detail may be had by an improvised method. A known point is obtained by fastening a short piece of lead wire vertically to the skin at the orbital margin exactly below the pupil. The distance from the upper end of the wire can be measured to various bony points for reference. The exposures are made from two points.

One method is to take one negative in the sagittal plane of the head and the other in the frontal plane. Another method is to parallel the side of the nose for one exposure and the temporal orbital wall for the other. During the process the patient's head or eye must not move. Not all penetrating substances are opaque to the x-ray. Because of the bony background it is sometimes difficult or even impossible to identify a very small foreign body. The magnet and the x-ray together constitute the most dependable methods of investigation.

The prognosis where foreign bodies enter the eye is bad. Some eyes escape serious consequences even though the foreign body is retained. Others demand prompt enucleation. Traditionally, the continued presence of a foreign body in the eye is potent for the excitation of sympathetic ophthalmia. This belief has been modified by accumulating reports of foreign bodies retained for many years without inflammatory symptoms in either eye.

It is reasonable to believe that injury to or irritation of the uveal tract, continued over a period of time, might excite sympathetic inflammation. There is no reason to anticipate iridocyclitis as long as the foreign body remains quietly in the vitreous, lens or retina, but signs of sympathetic disease should be watched for in every intra-ocular injury.

Loss of vision or loss of the globe depends less on the mere presence of a foreign body, with the possible exception of copper, than on the effects of

traumatism and infection. Since these factors are important, or essential, to the production of sympathetic ophthalmitis, both eyes are in peril. The removal of a foreign body in a case of recent injury does not lessen the extent of the traumatism, but may add to it, although magnet extraction rarely does so; it does not lessen the prospects of infection. Two factors are to be considered: The foreign body may irritate the tissues, or the eye may tolerate it.

A foreign body that irritates the tissues causes either a hyperemia or a proliferation of connective tissue. Hyperemia invites endogenous infection; connective tissue by contraction or shrinking perpetuates the irritation. When the eye is intolerant of a retained body, the symptoms are those of irritation plus a peculiar nervous reaction that resembles a traumatic neurosis; there is little or no hyperemia, infection, or proliferation of connective tissue.

The eye may tolerate a foreign body quite well, especially if vision, more particularly central vision, is good. Toleration must not be confused with immunity. An imbedded or encysted foreign body can work loose after many years and cause recurrent attacks of pain with symptoms of irritation; yet cases are not infrequent in which the eye remained quiet and was useful during the life of the patient.

The cases that have done best are those in which the injuring body was a small, sterile, nonmetallic one that damaged vision very little, and that was imbedded in the natural tissues or became permanently encysted or encapsulated by newly formed tissues. A conservative attitude is advised as long as vision is good and the eye is free from pain and hyperemia. Loss of vision alone is not an indication for enucleation.

Vision diminishes to total blindness in so many cases that this outcome must be expected in every case. The blind eye should be removed when there is evidence of inflammation or degeneration in it, or when the uninjured eye shows signs of sympathetic inflammation. Detachment of the retina in a blind eye with a retained foreign body leads to atrophy; these conditions as well as iridocyclitis in a shrunken eye are indications for enucleation.

In deciding on the management of any case, due consideration is given all of the factors. In a recent case the treatment may depend upon the extent of the traumatism and the probabilities of infection. The size, shape, surface, nature, chemical properties, and position of the foreign body, and whether it is free or imbedded must be considered.

A small smooth round object of lead or glass well imbedded causes far less apprehension than a rough fragment of iron or copper that is free to move about the interior of the eye. The latter may become encapsulated after a period of inflammation, but it cannot be known how soon it may excite a recurrence of the symptoms.

Treatment is decided upon several factors: (1) Accessibility, the location of the body; (2) duration, the time it has been in the eye; and (3) its magnetic or nonmagnetic nature. Three questions may be asked: (1) Is the vision good? (2) Will attempted removal cause more damage than already exists? (3) Is infection probable? The rule is to remove the body if it can be done without added damage to the eye; the earlier it is done the better the prognosis.

Where the foreign body is in the anterior chamber, its removal should be attempted under local anesthesia before dilating the pupil. There is then a broader surface of iris to prevent the body from slipping back into the eye, and the iris is not thrown into folds in the periphery to entangle the body.

A keratome incision is made in the cornea parallel with the plane of the iris and as near it as possible. The incision on the posterior surface is narrower than that on the anterior surface (Fig. 153); the cut in the posterior surface determines the size of the opening through which the body is to be removed. In withdrawing the keratome the minimum amount of aqueous is allowed to escape. Fine forceps or the magnet is used to extract the particle. When it is entangled in the iris, that structure may have to be drawn out of the wound; the part containing the body is snipped off with scissors. Aseptic dressings are required until healing is complete.

Fig. 153.
Compare with Figure 172.

A foreign body in the lens almost invariably causes cataract. When the magnet will not extract the particle it had better be left temporarily as it is. When the lens cortex is not absorbed, the cataract will ripen and may be removed; the foreign body will be removed with it. When the cortex has been absorbed, the body will be liberated and its removal can be undertaken. Such an eye should be watched; when the lens substance swells an intumescent cataract forms, and secondary glaucoma follows. Surgical treatment in the presence of this complication may be attended by intra-ocular hemorrhage or detachment of the retina.

In general, the forceps or magnet removal of a foreign body in the vitreous may be attempted through the original wound if it is still open, or through a surgical wound made for the purpose.

The deliberate opening of the sclera for the purpose of removing a foreign body is not endorsed by all surgeons. Such procedures have a reputation for an unhappy ending, although this cannot always be attributed to the operation. The incision should be made anteroposteriorly, not equatorially. The region of the ciliary body or the danger zone must be avoided. The magnet or the forceps may be employed. Nonmagnetizable substances offer the greater difficulties. It is often better to leave them alone than to add to the injury.

Damage to the vitreous by the entrance of the foreign body or its removal frequently results in the formation of fibrous bands which cause detachment of the retina. An infection is likely to terminate in panophthalmitis. A rapidly developing plastic iridocyclitis usually demands immediate removal of the eye to avoid sympathetic ophthalmitis.

Magnet extraction is indicated for magnetizable foreign bodies. When a case is seen very early this method is applicable without waiting for a roentgenograph. The body very rapidly becomes firmly imbedded in exudates or hemorrhage, and its subsequent removal becomes increasingly difficult. However, an x-ray localization may protect the eye from unnecessary manipulation. The magnet should be tested in each instance before applying it. The pupil must always be dilated.

Electromagnets are of two kinds according to size. Haab's giant magnet is very powerful. The hand magnets, such as Lancaster's, are relatively weak, but have a definite place in this work. The latter may be strong enough to remove a foreign body; very frequently they are the instruments of choice with which to complete the removal. Ring magnets are also used by some surgeons. All forceps, scissors and other instruments to be used in the magnetic field should be made of nonmagnetic metal. Steel that includes combinations of chromium or aluminum is nonmagnetic. Metallic foreign bodies of this material cannot be removed by the magnet.

Fig. 154.

After surgical preparation and cocain anesthesia the magnet may be applied to the entrance wound or to a surgical opening. The current is turned on. If it induces pain, the foreign body is magnetic. The rapid closing and breaking of the circuit is of value in dislodging an imbedded foreign body.

Most surgeons prefer a corneal point of attack. The tip of the magnet is brought into contact with the center of the cornea and the current is turned on as before; the circuit is rapidly opened and closed if desired. After the foreign body is brought into the anterior chamber it is extracted. When it is brought against the posterior surface of the iris that structure will bulge. The magnet tip is held so that the direction of its force will be in line with the bulging part of the iris and the pupil (Fig. 154); the body may then be drawn into the anterior chamber.

The magnet tip may be applied to various portions of the cornea successively, or to the sclera, but never over the region of the ciliary body. The foreign body may be removed from the anterior chamber in the manner already described. The escape of too much aqueous may require waiting for the anterior chamber to re-form.

Jackson devised a method and a special scissors of magnetizable metal for

the removal of magnetizable bodies that are firmly imbedded or encysted, or that resist efforts to remove them otherwise. The instrument is introduced into the eye toward the object, thereby making a new passage along which it may be removed. When the body is reached, a few snips of the scissors may loosen whatever tissue is retaining the body in its situation. The magnet is then connected with the scissors, which are withdrawn. The procedure may be repeated as required.

It sometimes happens that the foreign body is pulled off the magnet tip by the lips of the wound. A fine forceps may be inserted at this point; it will serve to dilate the wound and grasp the object.

Many attempts to recover foreign bodies are fruitless. Many eyes are lost even though the body has been removed. The prognosis in magnet extraction should be guarded.

As foreign bodies entering the eye may have been contaminated with tetanus bacilli, it is appropriate to administer antitetanic serum in these cases.

The after care of foreign body cases is that of postoperative care when the globe is intentionally opened for any purpose.

Since watches are readily magnetized, they should be laid out of the magnetic field during the operation.

CHAPTER XIV

GLAUCOMA

ESSENTIALS OF THE ANATOMY

Glaucoma is a term used to designate the ocular manifestations of any disease in which the principal local signs and symptoms depend upon an elevation of intra-ocular pressure with a consequent increase in the tension on the coats of the eye. Clinically, two types exist: The primary form is independent of any demonstrable antecedent disease of the eye; the secondary form is dependent upon some antecedent ocular disease. Glaucoma is also called hardening of the eyeball, dropsy of the eyeball, hydrophthalmos or juvenile glaucoma, and buphthalmos or infantile glaucoma.

The suspensory ligament of the lens is composed of fibers that arise from the ciliary body and end in the peripheral portions of the anterior and posterior surfaces and the equator of the lens. The aqueous humor is a lymphlike liquid supplied from the epithelium of the ciliary body and processes. A small part of it goes into the vitreous. The principal portion accumulates in the posterior chamber, passes through the pupil into the anterior chamber, and drains through the spaces of Fontana and the canal of Schlemm into the anterior ciliary veins. A small part probably enters the crypts of the iris, whence it may find its way into the suprachoroidal space.

The posterior chamber is bounded anteriorly by the iris, posteriorly by the suspensory ligament and the periphery of the lens, and circumferentially by the ciliary body. The anterior chamber is bounded anteriorly by the cornea, posteriorly by the iris and lens, and circumferentially by the pectinate ligament. The pupillary border of the iris rests on the lens. Aqueous can pass from behind forward but not from before backward under ordinary conditions.

The pectinate ligament is composed of a system of fibrous meshwork which is apparently continuous from Descemet's membrane. The fibers are inserted into the root of the iris and into the anterior part of the ciliary body. The arrangement of the fibers defines the spaces of Fontana. These spaces are modified in size and shape by accommodation and by contraction and dilatation of the pupil.

The endothelium of Descemet's membrane is continued over the fibers of the pectinate ligament; it covers the anterior surface of the iris, but does not

enter the iris crypts. The endothelium excludes aqueous from tissues bounding the anterior chamber, except as noted. Thus the optical integrity of the cornea is maintained.

The angle formed by the iris and cornea is called the angle of the anterior chamber, the iris angle, or the filtration angle. The canal of Schlemm is an intramural system of communicating spaces in the sclera near its junction with the cornea. Schlemm's canal is called the sinus venosus sclera (B.N.A.), but whether it carries venous blood or lymph is not decided.

Concerning the lymph system of the posterior part of the eyeball, comparatively little is known. The suprachoroidal space is in communication with the tissues investing the venæ vorticosæ. Tenon's space has not the characteristic structure of a lymph passage. The intervaginal spaces about the optic nerve are lymph passages. The hyaloid canal is a remnant of a fetal structure and varies greatly in different eyes; it is not a proven lymph passage.

The significance of a posterior drainage is inferred by the variation in the depth of the anterior chamber in different glaucomatous eyes. In buphthalmos the anterior chamber is often very deep because of a congenital failure to develop the drainage system through Schlemm's canal. In senile patients with glaucoma the anterior chamber is usually very shallow because changes, possibly sclerosis, have damaged the posterior drainage system, and the anterior changes follow the primary process. In some of the senile cases the anterior chambers are deep; this variation supports the theory of anterior and posterior drainage.

METHODS OF PRODUCTION

Glaucoma only exists when the production of aqueous exceeds its drainage, or when drainage falls below production. Production may be normal and drainage diminished, or drainage may be normal and production increased.

So far as is yet known, glaucoma must be ascribed to a failure in the drainage mechanism, and not to a simple increase in the production of aqueous. The latter doubtless occurs, but for very brief periods only.

Glaucoma is divided clinically into primary and secondary forms. The first type is subdivided according to the rapidity of onset. When this is sudden the eye has no time in which to adjust itself to the increased tension; it is called congestive or inflammatory because it is accompanied by signs of congestion or inflammation. It may be acute or chronic. When the onset is slow, the tissues become adjusted to the gradually increased tension and there are no signs of congestion or inflammation; this is called noncongestive, noninflammatory or simple glaucoma. It is always chronic. The secondary type depends upon some antecedent eye disease. It may be acute or chronic.

SECONDARY GLAUCOMA

As primary glaucoma comes on without a recognized cause, while the secondary form affords the advantage of knowledge of the antecedent condition and the history of the case, a better understanding of the changes that occur in glaucoma may be obtained by first studying known facts. For this reason secondary glaucoma is considered first.

In the conditions about to be enumerated, inflammation and venous stasis occupy a prominent position. When the cornea is perforated the iris floats into the wound and unites with it in healing. Healing is accomplished by a process analogous to inflammation. Contraction occurs in the cicatricial tissue and the iris is drawn forward against the cornea for a part of its circumference. Where a considerable extent of the filtration angle is sealed in this manner, so that the escape of aqueous is greatly hindered, glaucoma will result.

The anterior chamber is opened for the extraction of cataract; when iris tissue or lens capsule becomes incarcerated in the wound, an inflammatory reaction may cause the occlusion of the angle of filtration to an extent that drainage is diminished. Epithelium may grow into the wound and form a cyst of the anterior chamber, and the aqueous cannot pass through the epithelium; drainage is stopped.

Injury to the lens may cause the cortex to swell and crowd the iris against the cornea in the filtration angle. Needling of an after-cataract interferes with drainage when lens material clogs the filtration spaces, or where vitreous enters the anterior chamber.

Dislocation of the lens may cause glaucoma. Where it is displaced into the space between the ciliary processes and the iris it will crowd the latter against the cornea, while on the opposite side of the pupil the vitreous will similarly crowd the iris against the cornea. Where the lens is dislocated into the vitreous, inflammatory reaction with changes in the aqueous can cause glaucoma; when it has lodged in the pupil no aqueous can pass through. When the lens occupies the anterior chamber it practically fills that space and diminishes outflow. The continued production of aqueous crowds the iris against the lens and no fluid can pass the pupil.

In diseases of the uveal tract, particularly iridocyclitis, and more especially when the inflammation is of the plastic type, glaucoma is a frequent complication. The products of inflammation, round cells, pigment-cells, albumin and fibrin clog the spaces of Fontana. This exudate readily undergoes organization and permanently seals the filtration spaces. Also, the swollen iris is crowded by the swollen ciliary body against the angle of filtration and no fluid can enter the canal of Schlemm.

Posterior synechiæ and pupillary exudates or membranes prevent the flow of aqueous into the anterior chamber (see Figs. 130 and 134). Whether iris bombé forms or not, the periphery of the iris is pushed against the cornea and the escape of aqueous is impeded or prevented. The synechia may be annular or total. A foreign body in the eye may excite an inflammation, and the products of the inflammation, as in uveal reactions, may occlude drainage by the accumulation of débris in the angle of filtration.

Intra-ocular tumors cause secondary glaucoma in practically every instance. The composition of the fluids is changed, so that tumor cells and débris may occlude drainage. The tumor can make mechanical pressure against one or more vortex veins as well as against the lens and iris. Retention of fluid and elevation of tension result.

Intra-ocular hemorrhage from rupture of a vessel, thrombosis of the central vein, albuminuric retinitis or other conditions can change the composition of the fluids within the eye. An iridectomy or any surgical procedure which would suddenly lower the tension might increase the hemorrhage. Miotics do little or no good, as the modified fluid does not flow through the spaces of Fontana, no matter how widely open they may be.

High grades of myopia, and sometimes choroiditis, are productive of glaucoma because of changes in the composition of fluids, or because of some change in the filtration system in the posterior part of the eye. Staphylomata of the cornea and sclera require certain adhesive changes in the iris or ciliary body for their production (see Plate XII). These changes occasionally bring about alterations in the intra-ocular fluids, and sometimes cause increased pressure.

A summary of the usual causes of secondary glaucoma attracts attention to several pertinent facts: The fluids of the eye are modified by inflammation and by venous stasis; pressure may cause venous stasis; pressure is directed toward the iris and the filtration angle except in cases of choroiditis, myopia, and in the early stages of some intra-ocular tumors where the posterior drainage system is affected. Due either to pressure or to altered composition of fluids, drainage of aqueous is inadequate in proportion to its production. This inadequacy in practically every important instance is located in the filtration angle; Fontana's spaces are clogged by débris, or are covered over by the iris. Aqueous is retained and intra-ocular pressure is raised.

PRIMARY GLAUCOMA

Certain observations made on a large number of glaucomatous eyes have given rise to a belief in an element of predisposition to glaucoma. The age incidence is mostly after forty; women are more frequently attacked than men,

and women are more subject to venous congestions. Hereditary influence is noticed in some families where the symptoms occur in consecutive generations from both fathers and mothers; each succeeding generation is affected at an earlier age, and this is called "anticipation." Racially, Jews are said to be more frequently affected.

Senility appears to be important. The anterior chamber is shallow. Sclerosis of the pectinate ligament means that the fibers are stiffened and thickened and that the spaces of Fontana are smaller. These individuals are subject to cataract, and the early stage of cataract may be accompanied by intumescence or swelling of the lens with pressure against the iris; progressive and rapid loss of accommodative power is a warning of the probability of glaucoma.

The hyperopia that is due to a small eyeball furnishes significant speculations. The ciliary muscle in a hyperopic eye is well developed. The small eyeball has a correspondingly small cornea, and a comparatively small circumlental space. The anterior chamber in a small eye is shallower than in a large one. Both eyes of a pair are usually of the same size, and primary glaucoma is bilateral, even though one eye may be affected before the other. The lens continues to grow throughout life. Lenses are rather uniform in size regardless of the dimensions of different eyes.

In the small eye, a slight congestion of the ciliary processes can bring them into contact with the periphery of the lens and thus obliterate the circumlental space. The lens is crowded against the pupil so that the flow of aqueous is arrested. Then the iris is pressed against the cornea and the aqueous cannot escape by way of the canal of Schlemm. Although relief may be obtained, the experience is repeated until the iris is attached to the cornea permanently and filtration ceases. The anterior chamber is shallower than it was before.

Acute Congestive Glaucoma is characterized by three stages: The prodromal period, the active period, and the period of absolute glaucoma which may be followed by degenerative processes.

The prodromal period may or may not be recognized. The patient complains of periods of foggy vision, of dull pain in the eye or of the same side of the head, and perhaps of having noticed rings of prismatic colors or rainbow halos around artificial lights. These symptoms are all due to temporary increases in intra-ocular pressure. Excavation of the disk is not to be expected during this period.

Visual acuity is diminished because the media are cloudy, the pupil is somewhat dilated and inactive and does not permit a sharp image, insufficient blood reaches the retina, and pressure on the retinal nerve-fibers affects conductivity. The cornea, when seen during an attack, appears steamy. This is more noticeable in the central portion over the dark pupil; it may resemble intersti-

tial keratitis, in which the cornea is hypersensitive. In glaucoma the corneal sensitiveness is diminished or absent.

Rainbow halos around artificial lights are related to the steamy cornea. Innumerable fine droplets of fluid (Fig. 155) in the edematous cornea diffract light, and the edema disturbs the normal relations of the corneal lamellæ and helps to cause the play of prismatic colors. The pain in the eye or head is generally neuralgic in character. The pain often radiates over the distribution of the trigeminus and distracts attention from the eye, especially if vomiting is present.

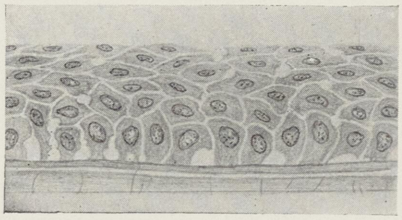


Fig. 155.

The pupillary dilatation is due to pressure on the ciliary nerves as they pass between the choroid and sclera, and to poor vision. For this reason the pupil reacts sluggishly. The lens may have a peculiarly greenish appearance. The anterior chamber is shallow. The tension is increased. Pressure within the eye diminishes venous drainage by way of the venæ vorticosæ, and the blood is directed toward the anterior ciliary veins which are engorged (Fig. 156). There is apt to be some injection of the circumcorneal vessels.

The usual history is one of recurring attacks of increasing frequency and severity. The pressure on the ciliary nerves and on the ciliary muscle weakens the power of accommodation rather rapidly, and so stronger and still stronger convex lens additions are required for reading or near work. Latent hyperopia often becomes manifest.

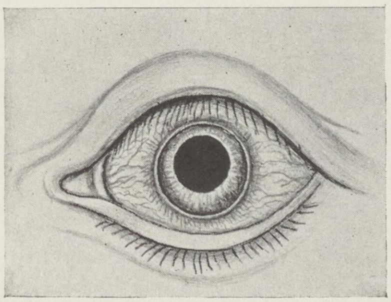


Fig. 156.

The attacks are prone to occur at night if there is insomnia; they come on after a period of emotion such as anxiety, worry, grief or mental excitement, with fatigue, alcoholic intoxication or indigestion, during periods of venous congestion such as menstruation, and after the use of mydriatics. Attacks have been known to follow prolonged bandaging of the eye or a prolonged exposure to darkness; in either case dilatation of the pupil may have occurred passively. With a dilated pupil the iris thickens and so diminishes the space in the angle of filtration, particularly where the anterior chamber is already shallow.

In *the active period,* an attack comes on without any other warning than what may have been given by recognized prodromal symptoms. Many attacks occur at night. One of the exciting factors already enumerated has been operative, although it may not receive credit unless properly associated with subse-

quent events. Vision is reduced; when the visual field can be taken it will be found contracted, more especially to the nasal side.

Pain is a prominent symptom. It affects the eye and radiates over the side of the head. The consequent headache may mask the real situation of the pain in the eye and obscure its significance. Lacrimation may be present. The pain frequently causes nausea and vomiting; there may be fever and general depression. These signs tend to further confuse the situation.

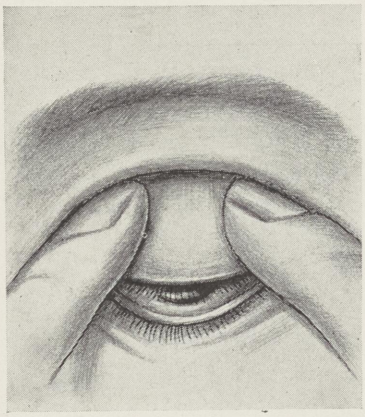


Fig. 157.—Taking Tension; Position of Finger Tips.

Tension is raised. This is determined by an instrument called the tonometer, which is equipped with a pointer and a graduated scale. Schiotz, Gradle's modification of Schiotz, McLean, and Bailliart instruments are available.

An approximation of the tension can be obtained by the finger test. The examiner stands in front of the patient, who is directed to look down. The tip of the forefinger of each hand is placed, one on either side of the insertion of the superior rectus muscle, 1 or 2 centimeters apart (Fig. 157), with the nails resting against the inferior surface of the bony orbital rim (Fig. 158) and the fleshy tips of the fingers in contact with the upper lid immediately over the equator of the eyeball where the sclera is thinnest.

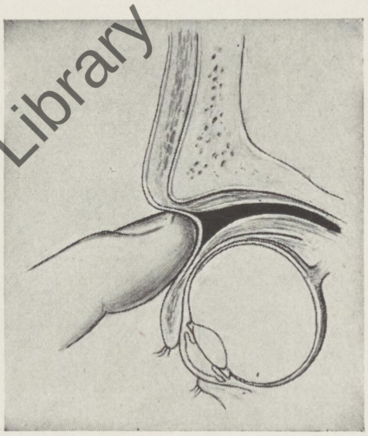


Fig. 158.—Taking Tension; Position of Eyeball.

By gently pressing alternately with the fingers, the eye with normal tension will be felt to dimple slightly when pressed upon. The hard eye will not dimple, but will appear to rock from side to side as pressure is made with first one finger and then the other. An alternate method is to hold or steady the globe with one finger and test for dimpling with the other. Tension above normal means glaucoma.

During an attack the lids are occasionally edematous or swollen, and there may be a chemosis or congestion of the bulbar conjunctiva. A history of recent erysipelas will prove confusing unless attention is paid to other signs of glaucoma. A zone of 2 or 3 millimeters width, the circumcorneal zone, may be colored a dull red or violet from congestion of the deep vessels about the cornea. These vessels resemble a fine fringe since they do not anastomose. In the same

area somewhat larger vessels may be engorged; these are the episcleral veins. The largest posterior conjunctival vessels are usually engorged also.

The intra-ocular pressure diminishes or prevents drainage by way of the vortex veins. The blood must then flow forward, and as a consequence the anterior ciliary veins are markedly distended. The enlargement of these veins is always suggestive of glaucoma.

The cornea appears steamy; as a rule this opacity is uniform during a glaucomatous attack. Corneal sensation is diminished or lost. Punctate deposits may appear on the posterior surface of the cornea; the aqueous may be turbid. Because of these conditions no ophthalmoscopic examination is possible. The anterior chamber is shallow.

The iris is congested early, but pressure soon brings on an ischemia. The iris loses its distinct pattern and becomes dull and discolored or grayish. Because of pressure or of anemia the nerves to the sphincter of the pupil are partially or wholly inhibited. The pupil is dilated, usually oval, with the long axis vertical. It contracts sluggishly or not at all to light. In iritis the pupil is contracted and often fixed to light; in many other respects iritis may be confused with glaucoma. A table on differential diagnosis is given under the subject of iritis (see page 153).

The principal signs of glaucoma are the steamy cornea, the dilated and fixed pupil, and increased tension. When all of these are absent it is well-nigh impossible to diagnose glaucoma except with the ophthalmoscope.

After days or weeks of fluctuation in the severity of the symptoms there is usually a remission which lasts for an uncertain period. The second attack can be so long postponed that the patient may believe he has been permanently relieved. In the usual course of events, without treatment, subsequent attacks are inevitable. It is possible that the first attack may not entirely subside. In either case glaucoma becomes established.

Successive attacks each leave the eye in a worse condition than it was in before. The reduction of vision and the contraction of the visual fields are progressive. Objective signs do not entirely disappear. The tension rarely goes back to normal except temporarily. The power of accommodation is reduced. The increased intra-ocular pressure sometimes increases the equatorial diameter and shortens the eyeball; hyperopia results.

When glaucoma is found in one eye the other must be examined also. It is possible that the second eye may have been the subject of a glaucoma that was unrecognized or that has been forgotten. Or the attack may have been of the noncongestive type where the only symptom was a reduction of vision, but where structural changes are demonstrable later on. Noncongestive glaucoma confers no immunity from the congestive form.

During the interval between attacks, or during the periods when the media are relatively clear, an ophthalmoscopic examination may be made, the blind-spot and scotomata may be plotted, and the visual fields may be mapped out.

Absolute Glaucoma, the third stage, is a consequence of repeated attacks. The vision fails to recover any loss during the remissions, or there may be no remissions. Blindness finally becomes absolute. Tension is constantly above normal and the globe is hard, even stony hard. Appearances of congestion subside, but there remains a dark-colored violet ring around the cornea because the anterior ciliary veins are engorged. Pain is constant, periodical, or absent. The patient stares into vacancy characteristically; the eyes look at nothing.

The cornea may continue to be steamy or it may become transparent, but it does not regain sensitiveness. The anterior chamber is shallow and sometimes it is almost obliterated. The iris is atrophied, thin and narrow; it is usually gray. The posterior layers of the iris are derived from the retina; they do not atrophy like the stroma and anterior layers. As the latter retract they draw the pigment layer into the pupil so that it is bordered by a black ring. This is called an ectropion of the pigment layer. The pupil is widely dilated and fixed to light; instead of a normal black appearance it looks gray or green on account of the changes in the lens.

Fundus changes continue. The glaucomatous excavation or cup deepens, and the disk shows atrophy; it may be surrounded by an atrophic halo peculiar to glaucoma. Changes in the vessel walls, altered secretions, disturbed nutrition and degenerative processes are prone to occur. These become manifest in all of the ocular tissues; they are progressive and often lead to destruction of the eye.

The cornea is sometimes glassy, but is usually cloudy. Its tissues have poor resistance; keratitis may develop; ulceration with subsequent perforation is not uncommon. Such an accident leads to iridocyclitis or to panophthalmitis. The continued high pressure weakens the sclera, and localized bulgings or staphylomata form. A weakened zone between the iris and ciliary body is followed by an intercalary staphyloma; when it occurs in the ciliary region, a ciliary staphyloma results (see Fig. 120).

Equatorial staphylomata are formed because the equatorial region of the sclera is its thinnest and weakest part. The venæ vorticosæ ordinarily leave the eyeball between the recti muscles. Changes in the tissues about the exits of the veins weaken those parts and bulging occurs. When staphylomata are formed between each of the muscles the eyeball tends to become square. Staphylomata are recognized by their bluish color, which is caused by the uvea showing through the thin and almost transparent sclera. Spontaneous rupture of the globe may take place.

Cataract may form. Instead of the dense white of the senile cataract, that due to glaucoma is of a bluish tint, has a silky lustrous surface, and appears to be swollen. This may form while vision is otherwise fairly good. Extraction of such a cataract must not be attempted; in the presence of increased tension the necessary corneal incision evacuates the aqueous rapidly with consequent sudden reduction of the pressure, and intra-ocular hemorrhage is likely to ruin the eye. As a wide peripheral iridectomy is a proper treatment for glaucoma, this should be made. If, after the lapse of a month the projection is good and the tension down, an attempt may be made to remove the lens.

Choroiditis may form in a glaucomatous eye. Detachment of the retina occasionally follows. During the process of detachment there may be subjective sensation of light, mechanically produced by intermittent separation, and the patient is falsely encouraged in the hope of returning sight. Tension is lowered in retinal detachment.

Atrophy of the ciliary body occurs in the degenerative process. When this happens, little or no aqueous is produced and the tension falls below normal. As destruction of ocular tissues progresses, the globe softens and shrinks or atrophies.

Fulminating glaucoma is rare; it comes on suddenly and violently without the warnings of recognized prodromal symptoms. The reaction is so intense that it is rapidly destructive, and vision may be permanently lost within a few hours. It belongs to the congestive type.

Instead of entering the stage of absolute glaucoma, the acute form may take on the characteristics of the chronic form of the congestive type.

Chronic Congestive Glaucoma is not distinct from the acute form except in behavior. The chronic form is subject to acute attacks and the acute form may become chronic. In the chronic form the onset is gradual or insidious and the congestive attacks are milder. It may evade detection until permanent damage has been done. The prodromal stage may almost insensibly merge into the congestive stage. There may be no real congestive attack, such as is the rule in the acute form.

The local signs are those described for acute glaucoma, but they may be far less marked. Pain is usually less violent and is not continuous. Vision is lost so gradually that the patient is very often not perturbed about it, and continues to delay seeking medical advice until too late to save sight. In some cases central vision is fairly well preserved until late changes demand attention. This emphasizes the importance of mapping visual fields in doubtful cases. Nasal fields are more contracted than temporal, but there is a general outline of concentric contraction. The patient who has chronic congestive glaucoma may not observe halos about artificial lights. This form is bilateral, but one

eye is usually involved before the other. When found in one eye, the other must be kept under observation.

The acute and chronic forms have the same type of fundus and disk, and terminate in the same way by blindness and occasionally by degeneration of the eyeball.

The slow gradual course may lead to errors in diagnosis. Optic atrophy is apt to be confused with chronic glaucoma. The color fields contract more rapidly than form fields in optic atrophy, while all fields contract in proportion in glaucoma, with a more rapid contraction on the nasal side. The glaucoma patient detects light, light minimum or L.M. less efficiently, but detects changes in the intensity of light, light difference or L.D. more readily than does the patient with optic atrophy.

The slow course sometimes leads to a diagnosis of cataract. The lens in glaucoma looks gray, bluish or gray-green, rather than the dense cold white of a senile cataract. The fundus reflex is not demonstrable in cataract as it is in glaucoma.

In diagnosis the state of tension must not be relied upon, for the tension varies and may not be elevated at the moment of testing. The important points are the history of the case, the appearance of the cornea, the depth of the anterior chamber and the outline of the visual field.

Simple Glaucoma or noncongestive glaucoma has no marked external signs. The anterior ciliary veins may be moderately engorged and the cornea may be faintly cloudy at times. The pupil is normal and active, or a little dilated and sluggish. There are no congestive attacks and no pain. The principal complaint is the gradual loss of vision with periods of misty sight. Halos may or may not be observed about artificial lights. Accommodation is affected and presbyopia develops prematurely. Concentric contraction of the field progresses more rapidly on the nasal side. Central vision is preserved longer than peripheral vision; complete blindness ensues as a rule.

This type is of slow development and its progress may extend over many years. It is disposed to appear earlier in life than the congestive type, but it may change into the latter. At times the only objective evidence obtainable is by the ophthalmoscopic examination. The disk is always cupped, but in early periods the excavation may disappear while the tension is temporarily near normal. The depth of the cup depends upon the duration of the process. The glaucomatous halo usually surrounds the excavation in this type.

The blind-spot is generally enlarged, particularly above and below. Scotomata form in the fields, mostly in the vicinity of the blind-spot. Tension is variable; at times it seems normal. As a rule an elevation of pressure will be found when the tension is tested at different times during the day. Failing

sight with characteristic fields, cupped disk and scotomata stress the importance of frequent tonometric readings.

Juvenile Glaucoma Simplex belongs to the hereditary group, especially as it displays the "anticipation" feature. It occurs in childhood and youth. It is apt to affect several members of the same generation of brothers and sisters. In cases of painful eye with defective sight in children, particularly when there is a history of glaucoma in the family, the eyes should be examined with care before excluding glaucoma. This must be differentiated from buphthalmos since both have been called hydrophthalmos.

The juvenile type has many features in common with senile glaucoma, while buphthalmos has comparatively few.

Buphthalmos or "ox eye" appears congenitally or in infancy. It is characterized by a uniform enlargement of the globe in all dimensions, and all of the tissues of the eye except the lens are affected. Because of the enlargement the globe may appear to be proptosed. The sclera is thinned until it acquires a bluish color from the underlying uvea. The cornea is enlarged, and this is called keratoglobus or megalocornea; but the cornea can become enlarged with no demonstrable evidence of glaucoma. Faint parallel, branched or lattice-shaped lines on the posterior surface of the cornea indicate ruptures in Descemet's membrane; they look white by oblique illumination, and dark with the ophthalmoscope.

The anterior chamber is very deep. The pupil is dilated and sluggish. The iris becomes atrophic, thin and narrow. As the eye enlarges the iris is drawn away from the support of the lens; consequently it trembles (iridodonesis) when the eye or head is moved. The suspensory ligament is stretched so that the lens is drawn backward and thinned or flattened. These various changes produce a moderate myopia. The vision is apt to be poor otherwise, and nystagmus is not uncommon.

The disk is excavated; the depth depends upon the duration of the glaucoma. The course is slow and the prognosis is unfavorable, although the process may stop short of complete blindness. Buphthalmos may appear as one of the symptoms of von Recklinghausen's disease.

The cause of buphthalmos is due to a lack of drainage anteriorly. The fault may be effected in one of two ways: The filtration system may have failed to form properly, or an intra-uterine or early infantile inflammation may have occurred. Schlemm's canal may fail to form, Fontana's spaces may fail to open, or they may be obstructed by tissue or organized exudates, or an anterior peripheral synechia may form and occlude the filtration angle. The sclera yields uniformly to the pressure. Tension is as constant in buphthalmos as it is in the glaucoma of senile patients.

For differentiating buphthalmos and simple juvenile glaucoma Snell constructed the following table:

Feature	Buphthalmos	Juvenile Glaucoma
Onset	First decade	10 to 40 years of age
Direct inheritance	Rare; does not recur in succeeding generations	Always, and in consecutive generations
Corneal diameters	More than 12 millimeters Megalocornea	Less than 12 millimeters Microcornea
Anterior chamber	Very deep	Usually shallow
Type	Chronic	Acute or chronic
Color of sclera	Blue	Normal

CHANGES IN FUNDUS

The changes about to be discussed are the results of increased tension. It is not always possible to demonstrate them in early stages of glaucoma, for the evidence of an attack may disappear spontaneously or from treatment.

The Glaucomatous Excavation of the Optic Disk.—The corneoscleral junction and the lamina cribrosa are the weakest parts of the coats of the eyeball. The ophthalmoscopic appearances characteristic of glaucoma depend upon tissue alterations in and about the disk. These alterations are possible because of the weakness of the lamina. The lamina cribrosa is composed of a loosely woven tissue which bridges the scleral foramen. Through its spaces the nerve-fibers from the ganglion-cells of the retina pass out to form the optic nerve. The lamina is slightly curved, with its concavity toward the vitreous.

With pronounced intra-ocular pressure the concavity of the lamina is deepened. Where the pressure continues, the entire lamina or any part of it can be pushed back into the nerve sheath. There is a traction effect on both the blood-vessels and the nerves of the retina. The nerve-fibers are also subjected to extra trauma as they are drawn over the sharp margin of the rigid scleral ring.

The ophthalmoscopic appearance of the backward displacement of the lamina is that of a cup or excavation (Plate XI), the depth of which varies with the intensity and duration of the increased tension. Many normal disks contain a physiological cup which may be very deep. In no case does the edge of such a cup coincide with the margin of the disk, except that the surface of the disk may gently slope to its temporal border without showing a sharp bending or interruption of any retinal vessel. At least one vessel will be hooked over the edge of a glaucomatous cup.

The nerve trunk sheaths are of larger caliber than the scleral foramen. The foramen is wider at the outer surface of the sclera than it is at the inner

Plate XI.—Glaucomatous Excavation of the Disk.

surface. When the lamina is pushed behind the level of the sclera, it broadens or flattens out in the more capacious sheath of the nerve. Thus the glaucoma cup may have overhanging edges, and this gives rise to the appearance known as "excavation of the disk."

As usually pictured, the typical glaucoma cup is uniformly depressed. Such uniformity is not always present, for one part may be more depressed than another. Rarely a small segment of the disk margin will show a steep or abrupt offset. Where a retinal vessel crosses the margin at such a point, it will appear to be hooked over the edge of the cup (schematic Fig. 159). If the cup is of the excavated type, the vessel will wholly disappear beneath the edge. One hooked vessel justifies a search for glaucoma. More than one such vessel almost certainly means glaucoma.

An exception is found in cavernous atrophy of the optic nerve (*q.v.*). In this the fields for colors, light sense, and normal tension will be like those signs as found in optic atrophy, and unlike them as observed in glaucoma.

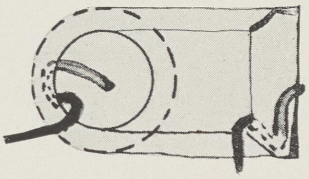


Fig. 159.

Not all glaucoma cups are excavated or even considerably depressed. A depression may be so shallow as to resemble the disk as seen in primary atrophy of the optic nerve. In either case many nerve-fibers will degenerate and produce stippling of the disk. In atrophy, the color of the disk is gray or white, while in glaucoma the color is likely to be pinkish because of hyperemia, and some edema may be present. The disk is white when it is definitely excavated.

A retinal vessel that disappears upon entering an excavated cup will reappear upon the floor of the cup; the visible continuity of the vessel is interrupted. A similar appearance is sometimes seen in a physiological cup, but there is no lack of continuity of the vessel, and the excavation does not extend to the margin of the cup. With the ophthalmoscope a weaker lens is required to focus a vessel on the floor of a cup than is needed for the same vessel at the level of the retina. By gradually weakening the lenses and increasing the distance, a vessel may be traced from the edge of the cup to its floor, provided that the edges are not overhanging. One or all retinal vessels may hook over the edge as in the eye of a cat.

The depth of the cup may be estimated by the parallax movement. By the indirect method a movement of the magnifying glass before the observed eye will seem to alter the relations of the edge of the cup and its floor. By the direct method the observer can develop the parallax by short movements of his own head from side to side. When he focuses on the margin of the cup, the floor will apparently move in the same direction in which he moves his

head. When he focuses on the floor, the margin will move opposite to the direction of his head movement (Fig. 160).

The parallax may be demonstrated on any cupped object, such as a wide-mouth bottle or an ointment jar. The same principles are used with the ophthalmoscope.

The depth of the cup may be measured with a fair degree of accuracy by the use of the direct method of ophthalmoscopy, provided the observer can suspend his accommodation. A note is made of the strength of the lens required to focus a vessel at the level of the retina. A similar note is made concerning the strength of the lens used to focus a vessel on the floor of the cup. The difference between the lenses is divided by three; the quotient is indicated in millimeters. If the first lens is a plus 1, and the second is a minus 2, the difference is 3, and the depth is 1 millimeter.

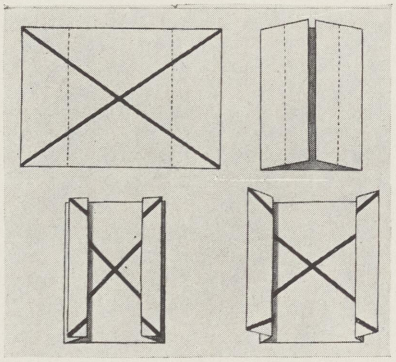


Fig. 160.
Fold paper along dotted lines to illustrate parallax.
(After Elliot.)

The traction exerted by the receding lamina sometimes seems to be greater on the nasal side, so that the retina appears to be dragged over that edge of the disk. In such cases all of the retinal vessels may seem to enter the cup on its nasal side.

In glaucoma the margins of the disk are usually sharply defined irrespective of the degree of cupping. This is particularly true in established cases where there has been some atrophy of the nerve-fibers with a resulting stippling of the lamina cribrosa. Frequently connective tissue is proliferated and new blood-vessels are formed. Such changes will conceal the stippling, and except for the new vessels, the disk may resemble that of secondary atrophy of the optic nerve.

A glaucomatous halo is sometimes found around the disk. The nerve-fibers from the retinal ganglion-cells nearest the disk pass through the periphery of the scleral ring. Thus they are brought into contact with the sharp edge of the ring and undergo degeneration from compression. The underlying layers of the retina atrophy, and the capillary layer of the choroid disappears. As the retinal pigment layer no longer conceals the larger vessels of the choroid, the halo is seen as a yellowish or grayish border about the disk. The halo fades into normal retina without a distinct boundary.

In some senile eyes a pale halo encircles the disk. No choroidal vessels are visible. This halo is comparatively narrow and its outer boundary is fairly distinct.

Increased intra-ocular pressure is responsible for several changes in the blood-vessels of the retina. Mention has been made of the formation of new vessels in the disk. A branch of a retinal vessel may effect an anastomosis with the choroidal circulation. Because of the congestion in the tissues of the lamina cribrosa, the retinal veins are often engorged, tortuous or varicosed in appearance. Some of the smaller arteries may be obliterated from the same cause.

Pulsation of the central artery of the retina is occasionally observed in glaucoma. It may be conspicuous or absent, for it varies with the tension. When intra-ocular pressure is almost as high as systolic blood-pressure, blood can only enter the eye by way of the central artery at systole. Pulsation is most readily detected at a bend of a vessel. Consequently, the pulsations of retinal arteries are best seen as the vessels bend to enter the cup. Pulsation can be induced by gently pressing against the eyeball with the finger tip. In glaucoma very little finger pressure is required to induce or to increase the pulsation.

Intra-ocular hemorrhages occasionally occur in glaucoma. They are usually linear, or flat rounded spots that radiate from the disk. Their presence means a guarded prognosis. Special precautions are taken before operating upon such an eye.

CHANGES IN FIELD

Visual field changes in glaucoma are due to diminished conductivity of the nerve-fibers of the retina. This is attributable to increased intra-ocular pressure. In one instance the finer blood-vessels of the retina may be obliterated, and conductivity is lost because of the resulting retinal ischemia. In another instance the nerve-fibers suffer directly from the pressure, and conductivity is lost on that account. Traction is a variation of pressure and has a similar effect on both vessels and nerve-fibers.

When ischemia is the cause of field changes, relief from the pressure will allow the vessels to fill with blood, and restoration of the field may be expected. Where the nerve-fibers have been damaged by the pressure they may undergo atrophy; it is improbable that the fields can be restored.

The degree of pressure is of less importance than the degree of tissue resistance in the patient. On this account the pathological effects of pressure vary greatly in different individuals. An eye with moderately increased tension may be permanently damaged, while another eye with greater tension may have very slight impairment. Healthy eyes vary in tension. So it is found that the relationship between tension and field changes is indefinite. The same uncertain relation holds good for the shallowness of the anterior chamber and for the degree of cupping of the disk.

The duration of glaucoma does not seem to depend entirely upon a persistent increase of tension. An eye may exhibit evidences of glaucoma and yet have little elevation of tension. The rapidity of onset and the intensity of the attack, modified by tissue resistance, determine the amount of damage.

The significance of visual field changes cannot be comprehended without a knowledge of the arrangement of the nerve-fibers and of their several courses from different regions of the retina toward the optic disk. It is helpful to imagine the retina as being spread out flat (Fig. 161). A vertical meridian and a horizontal meridian are drawn through the center of the disk. All of the fibers on the nasal side of the vertical meridian radiate like spokes from the disk. The horizontal meridian on the nasal side is unimportant.

The arrangement is complicated on the temporal side of the vertical line. The temporal quarter or third of the disk is distinctly paler than the remaining surface. This portion belongs to the papillomacular bundle of nerve-fibers which comes from the macular region. The fibers above and below this pale area supply the remainder of the retina on the temporal side. It seems then that these two bundles must be heaped up considerably above the common level as they pass over the margin of the disk. Possibly they protect the papillomacular bundle to some extent.

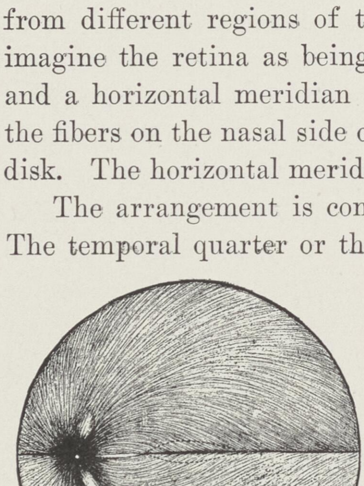


Fig. 161.—Nerve-Fibers. (After Weeks.)

Luciana was quoted to the effect that there are one million nerve-fibers, three million cones, and about twenty million rods in the retina. It is apparent that the average fiber is connected with three cones and about twenty rods. A fourth to a third of the fibers entering the disk come from the macular region; this is substantiated by microscopic examinations of cross sections of the nerve trunk immediately behind the eye (see Fig. 150). Each fiber which enters the pale part of the disk represents a single cone in the macular region, or perhaps a rod in the perimacular area. Each fiber outside this pallid area represents an indefinite number of rods and cones in the extramacular part of the retina.

The horizontal meridian which extends toward the temporal margin of the retina from the center of the disk helps to explain the field changes in glaucoma. The fibers from above and below the papillomacular bundle arch around the macular region, above and below respectively, and meet at this line, but do not cross it; this constitutes a raphé. These fibers are called the superior external and internal, and the inferior external and internal *arcuate* fibers (Fig. 162).

It is now seen that the nerve-fibers on the temporal side of the disk are much longer than those on the nasal side of it. The former not only follow the curvature of the eyeball, but they have an additional curve from their arcuate course. This arrangement avoids the disposition of unnecessary tissue in front of the macula, and yet allows the most uniform distribution of fibers possible under the circumstances.

It is generally believed that the shortest fibers lie deepest beneath the internal limiting membrane and supply the rods and cones nearest the disk; and that the longest fibers lie immediately beneath the internal limiting membrane and supply the rods and cones farthest from the disk.

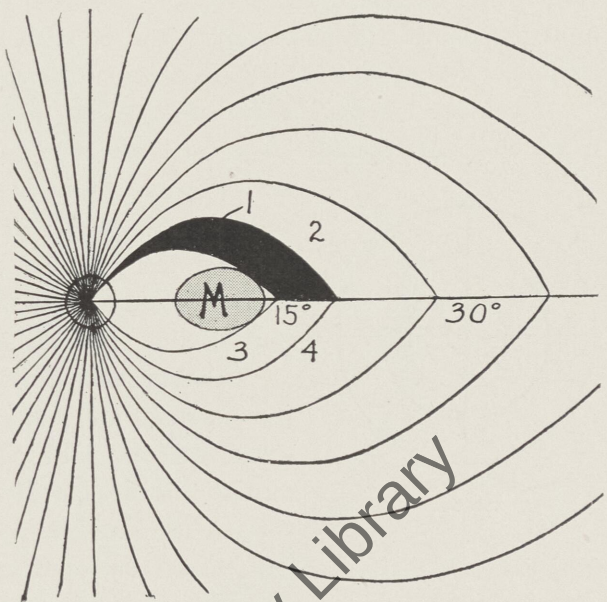


Fig. 162.—*M*, Macular Region; 1, 2, 3 and 4, Arcuate Areas. (After Elliot.)

When the fibers lying upon the sharp margin of the scleral ring are pressed upon, or are torn by traction as the lamina cribrosa is forced backward, the retinal elements from which these fibers are derived may undergo atrophy. Should these fibers come from that portion of the retina which immediately surrounds the disk, the glaucomatous halo is accounted for. The halo fades imperceptibly into unaffected retina, for the pressure does not seem to so profoundly affect those fibers which come from just a little farther out and which are not in actual contact with the hard edge of the scleral foramen.

The fibers which are heaped up above and below the pale temporal portion of the disk will be affected somewhat similarly. That is, the deepest will probably be more severely injured than those nearer the internal limiting membrane. For again, the deepest of them are most exposed to the sharp edge of the scleral ring, and come from retinal elements nearest the disk. Where these two bundles above and below the pale area are of equal thickness, or are equally affected by pressure, the earliest retinal degeneration will occur in those two spots adjoining the disk (see Seidel's and Bjerrum's signs).

Where these two bundles are unequal in size or are not equally affected by pressure, the degeneration will occur first on the more vulnerable side.

In either case, whether one or both sides are affected, the degeneration will be progressive along the arcuate course of either or both of these bundles. Thus it is possible for a ring of degeneration to entirely surround the macular region (see Fig. 162). This conclusion helps to explain the objective clinical findings connected with the blind-spot of Mariotte.

When these same prominent bundles are subjected to traction through backward displacement of the lamina cribrosa, or when the very long fibers of the bundles are inhibited by an increased intra-ocular pressure evenly applied, they cannot carry impulses from the rods and cones. Under such circumstances the longest fibers will likely be most profoundly affected, and thus the most peripheral retinal elements will be without communication with the brain centers. The effect of this is to contract the nasal field.

When one bundle is more incompetent than the other, the part of the retina to which it corresponds, upper or lower, will be affected, and a quadrant anopsia will appear in the field. Sometimes the nerve-fibers pass so evenly and uniformly over the margin of the disk, or the pressure or traction is so evenly applied, that the field will be contracted concentrically, or proportionately from all sides.

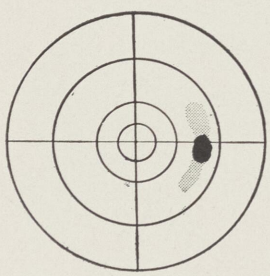


Fig. 163.—Seidel's Sign.

In other cases a whole half of the retina may become incompetent, and a hemianopsia will be found. In still other cases the field will show defects having an angular outline; in these, at least one angle is generally directed toward the fixation point. With the exception of the glaucomatous halo, none of these conditions can be detected with the ophthalmoscope. Changes in the visual field must be mapped and studied repeatedly.

The expression "enlargement of the blind-spot" needs explanation. The natural blind-spot cannot be enlarged, but the area of the blind-spot can be extended when the retinal elements surrounding the disk, or adjacent to it, are inhibited or degenerated. The methods for studying the blind-spot and the visual fields are given in the section on Visual Fields. Different-sized test objects are employed in making a diagnosis of glaucoma, and in recording its progress. Small ones of 1 or 2 millimeters are used for the blind-spot, while those of 2 to 10 millimeters are used for field work.

Seidel's sign demonstrates an extension of the blind-spot above or below, or both above and below (Fig. 163). It is one of the earliest field changes found in glaucoma. Where the extension follows halfway around the point of fixation, or to a point corresponding to the horizontal temporal raphé of the retina, it is called Bjerrum's sign (Fig. 164). This may be found above

Plate XII.—Corneal Staphyloma, Detachment of the Retina (Artefact) and Excavated Disk.

or below the fixation point, or completely encircling it. In both these signs the concavity of the extension is on the side of the fixation point.

Elliot's sign is a refinement of Seidel's sign; with a special instrument Elliot found that the extension of the blind-spot was pointed and not rounded.

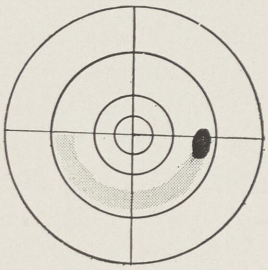


Fig. 164.—Bjerrum's Sign.

Rönne's nasal step is a term used to designate a field defect in which the blind area extends farther on one side of the horizontal line than on the other (Fig. 165). This finding indicates that the arcuate fibers are more affected on one side of the raphé than they are on the other.

It is possible for all of the retina to be affected except the macular region. The patient has tubular vision, in which the field can be represented experimentally by looking through two tubes. When this field is lost the eye is blind. Rarely, central or macular vision may be lost before the peripheral fields disappear; in such cases the papillomacular fibers are more vulnerable or less protected than in others.

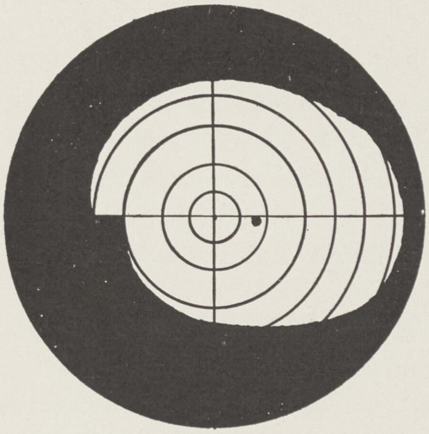


Fig. 165.—Rönne's Nasal Step.

Retinal adaptation to darkness is very slow in glaucoma. On that account, when a patient is brought from bright daylight into room light and immediately subjected to field study, the boundaries will be much smaller than if time had been allowed for adaptation. Alternatively, larger test objects will be required.

Elliot states his belief that the retinal arteries in a general way follow the course of the nerve-fibers, and that this factor may have some influence on field changes in glaucoma. No research has yielded positive conclusions on this point.

TREATMENT

The treatment of glaucoma is conducted along general, local, and surgical lines.

General treatment must be founded on the idea that primary glaucoma is the expression of some disease or constitutional disturbance in which the relationship between cause and effect has not been determined.

The highest possible state of health must be maintained. Constipation,

focal infections, and unhygienic habits must be corrected. Mental drains from grief, depression, worry, fatigue, exhaustion, excitement, and other emotions must be avoided. The patient can do much to build up resistance by having proper food at regular intervals, regular and ample rest especially at night, and fresh air at all times. He should be made comfortable. Alcohol, tobacco, and stimulating beverages should be used in moderation if at all.

Full doses of sodium salicylate have done good in many cases, notably in the so-called rheumatic group. Potassium iodid is indicated when arteriosclerosis is present. Salvarsan is of benefit to syphilitics. Hypnotics are necessary in sleeplessness, and chloral seems to be especially indicated in the insomnia of glaucoma. Morphin may be required for pain. In some cases purging and sweating are advisable.

In most cases of glaucoma the anterior chamber is shallow because the periphery of the iris is pressed against the cornea and drainage is obstructed. The pupil is dilated. Any means which will contract the pupil tends to pull the root of the iris away from the cornea so that drainage may be reëstablished. The crypts of the iris are more patulous when the pupil is contracted; they may possibly assist drainage.

The pupil is contracted in bright light, and the patient must not be kept in darkness unnecessarily. The pupil contracts with convergence as in reading or close work, and a reasonable amount of time spent in these occupations is beneficial unless contra-indicated otherwise. The pupil contracts in sleep; refreshing slumber should be encouraged. It is true that many attacks of glaucoma occur at night, but it is questionable if the patient has been comfortable and sleeping quietly.

Local treatment is instituted to maintain a functioning filtration system and to keep drainage active. The first object may be attained by contracting the pupil with miotics. Pilocarpin 1 per cent and eserin 0.25 per cent solutions are used. Pilocarpin is the less irritating but will have to be used oftener or in stronger solutions to equal eserin in effect. Eserin sometimes produces a conjunctivitis. Ordinarily, either solution is used in one drop doses one to three times a day.

Miotics cannot cure a glaucoma unless it is recent and has been caused solely by a mydriatic drug. They must not be depended on in progressive contraction of the visual fields or in sustained high tension. They are properly employed as an emergency measure during an attack, in preparation for operation, while waiting for a favorable time to operate, when for any reason an operation is contra-indicated, or when the patient declines operation.

"All forms of glaucoma are progressive and invariably end in blindness if allowed to go without treatment" (Swanzy). "Miotics merely postpone, they

do not cure" (Parsons). "If miotics have never cured a case of glaucoma, they have prevented many glaucomatous patients from being cured" (de Wecker).

Miotics are indicated in an acute attack of glaucoma. Eserin is usually employed as it is more energetic. A drop of 0.25 per cent solution should be instilled into the affected eye every ten or fifteen minutes for an hour, and then less frequently until the attack is controlled or until the pupil is contracted. As the second eye may be attacked also, or may be subject to glaucoma, it is advisable to instill a drop in it at the beginning of treatment. An atrophied iris will not respond to eserin.

The eyeball may be gently massaged through the closed lid for a minute or two. This often lowers the tension considerably. It should be followed by a drop of 5 per cent solution of dionin. This has a favorable effect on pain and acts as a lymphagogue. Then moist hot compresses should be used. Artificial leeches may be applied to the temple of the affected side. A pilocarpin sweat may do much good. Sweating must be employed cautiously in aged and decrepit patients.

When miotics have to be used over a considerable time, it is well to change from one to the other occasionally. Sometimes it is advantageous to combine them. Eserin is less apt to produce conjunctival irritation if fresh sterile solutions are used. A miotic is employed at night as the most favorable time, regardless of whether it is used during the day or not.

The second object of local treatment is to maintain active drainage. Dionin in 5 per cent solution, one drop once or twice a day, is advised. Fischer has advocated the use of sodium citrate in solutions of 4.05 to 5.41 per cent which are to be injected subconjunctivally in 0.3 to 1.0 cubic centimeter (5 to 15 minim) doses. These solutions may be combined with normal salt solution. Normal salt solution alone has been used in the same way. Massage and hot moist compresses help to promote drainage.

Surgical.—Ophthalmologists are agreed that surgical intervention is the only rational treatment for glaucoma. A large number of operations have been devised, but most operators prefer either iridectomy or trephining. Broadly considered, especially in the noncongestive type, an iridectomy is indicated where the anterior chamber is deep, while a corneoscleral trephine operation is proper where the anterior chamber is shallow or where there is much contraction of the visual fields. With restrictions, the same choice may apply in the congestive type.

The optimum time for operation is as soon as it can be done with safety after the diagnosis of glaucoma has been established. The operation should be done early during the quiet stage following a congestive attack, or at the

discretion of the surgeon when the case is of the noncongestive type. The pupil should be kept well contracted up to the time of opening the eyeball.

While the pupil is contracted the iris is taut, the anterior chamber is at its maximum depth, and there is more room in the angle between the iris and the cornea in which to work. The surgeon must not be misled by a false angle caused by an anterior synechia, or by the adhesion of the periphery of the iris against the posterior surface of the cornea. Miotics also tend to clear the corneal haze so that the operator has a better view of what he is attempting to do.

No operation should be undertaken without a search for infection of the tear sac. When the sac is found to be infected it should be extirpated, the canaliculi temporarily tied off, or the puncta sealed. In cases of uncertainty, one of the two minor operations is indicated. Infection of the conjunctival sac should be remedied before opening the eyeball. The patient is to receive such preoperative preparation as the surgeon desires. Some operators direct that the patient shall have a quieting dose of morphin and atropin or of hyoscin-morphin-cactin hypodermically before coming to the operating room. Chloral 0.65 gram (grains x) and sodium bromid 1.3 grams (grains xx) an hour before operating are usually adequate. Morphin may cause nausea and vomiting.

Local anesthesia is usually employed, and the choice between cocain, phenacain or butyn is personal with the operator. When a wide corneal opening is contemplated, it is advisable to infiltrate the branches of the facial nerve on the temporal side of the orbit to prevent squeezing of the lids. General anesthesia may be required for children and neurotic adults who are nervous, irritable, apprehensive, and generally unreliable in their behavior.

The *site* usually chosen for opening the eye is at the upper margin of the corneoscleral junction. A section of iris is removed in nearly all of the procedures; when this is made above the pupil it is of no visual importance so long as it is covered by the upper lid. Healing is better in this region because the conjunctiva extends farther over the cornea from above than elsewhere.

IRIDECTOMY AND ITS COMPLICATIONS

The instruments needed are: One eye speculum (Fig. 166), two fixation forceps (Fig. 167) without catches, a broad angular keratome (Fig. 168), a Graefe cataract knife, iris scissors (Fig. 169), blunt iris hook (Fig. 170), and an iris repositor or spatula (Fig. 171). The keratome is usually preferred, but when the anterior chamber is very shallow the operator may choose the cataract knife.

The patient lies prone on the table, and the surgeon stands or sits at the

head of the patient while the assistant stands at the shoulder or side. The lids are separated with the speculum. The fixation forceps is placed with the blades closed against the conjunctiva below the cornea at six o'clock. The blades are allowed to open while slight pressure is made against the eyeball. The blades are again closed, and in such a manner that they will pick up a vertical fold of conjunctiva. By a quarter turn of the forceps the fold is twisted, and thus affords a secure hold. The forceps must *not* press against the globe after the twist is made. The patient is then directed to look down, or the cornea is rotated downward with the forceps.

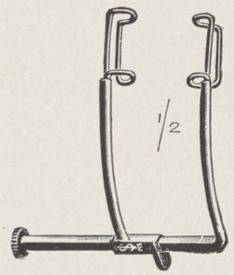


Fig. 166.—Murdock's Speculum.

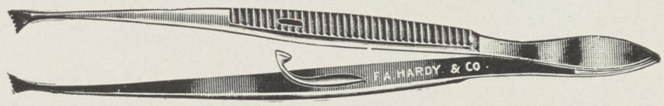


Fig. 167.

It is desirable to make the incision as near the anterior surface of the root of the iris as possible. This will vary between 1.5 and 3 millimeters back from the apparent margin of the cornea (Fig. 172). The point of the keratome is applied to the conjunctiva at twelve o'clock, and the plane of the blade is inclined to the surface of the globe at an angle of 45 degrees. Pressure

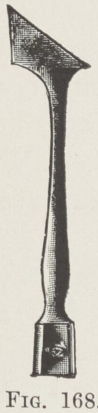


Fig. 168.

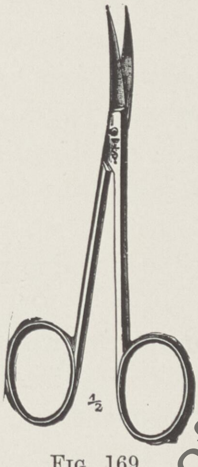


Fig. 169.

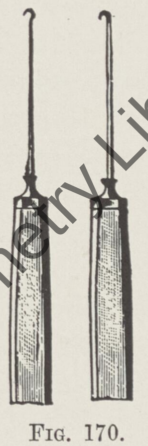


Fig. 170.

Fig. 171.

on the instrument must be in a direction parallel with the plane of the blade. When the point passes through the sclera and enters the anterior chamber, the handle is depressed so that the plane of the blade is parallel with the surface of the iris.

The operator watches for the point of the instrument to appear in the anterior chamber. Where it is properly introduced it will be bright; when it is

in corneal tissue it will look dull. If it fails to appear, it is probably *behind* the iris. Owing to the optical effect of the curved cornea, the point of the keratome will appear to be nearly 1 millimeter anterior to its actual position. This must be considered in operating.

Pressure within the globe forces the lens forward. The point of the keratome is directed well in front of the pupil to avoid injury to the lens capsule. At the same time it must be kept free from the cornea. In enlarging the incision the keratome is thrust first to one side and then to the other so that only one edge of the blade cuts at a time.

The external incision should be about 8 millimeters in length. The incision on the posterior surface of the cornea will then be about 5 millimeters long. Having completed the incision, the point of the knife is pressed toward the cornea before withdrawing the blade. The instrument is removed from the eye slowly to guard against a sudden escape of aqueous.

The operator may elect to open the anterior chamber with a cataract knife. The eyeball is held with the fixation forceps and the cornea is rotated downward as before. The point of the knife is applied to the conjunctiva in the same zone as directed for the keratome, but at seven minutes before twelve. The puncture is made by thrusting the knife toward the pupil, with the plane of the blade parallel with the plane of the iris.

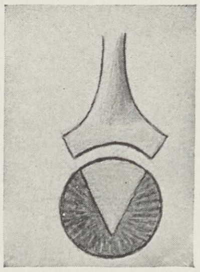


FIG. 172.
Compare with Figure 153.

As soon as the point is seen in the anterior chamber the handle of the knife is lowered until it is parallel with the horizontal meridian of the cornea. The blade is then worked across the anterior chamber in the direction of seven minutes after twelve, where the counterpuncture is made. The edge of the knife is kept as close to the iris as possible without wounding it. The knife cuts its way out by a sawing motion and without undue pressure against the tissues ahead of the edge.

When the sclera has been divided, the conjunctiva will raise over the knife edge. The latter is then turned forward and the conjunctiva is divided by a sawing movement. This makes a small conjunctival flap. The incision in this case will be comparable in position and extent to the one made with the keratome. It should be slightly less than one-fourth the circumference of the cornea. When the incision is accurately made, the section lies at the same distance from the cornea at all points.

The section having been made, the assistant takes the fixation forceps while the surgeon takes the iris forceps in his left hand and the iris scissors in his right. The closed iris forceps blades are introduced into the anterior chamber by gently depressing the posterior lip of the wound. When near the pupillary

margin of the iris the blades are allowed to open. The iris will probably bulge up between them so that it can be grasped without pressure against the lens. Slight pressure against the posterior lip of the wound is helpful.

Where the iris tissue is atrophic and fragile the forceps will likely tear it. When this happens or if for any other reason the iris cannot be caught with the forceps, it will be necessary to use the iris hook. It is inserted flatwise into the anterior chamber and then turned to engage the free border of the iris. Extreme care must be exercised to avoid injury to the lens.

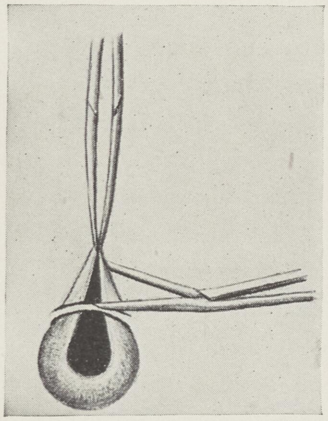
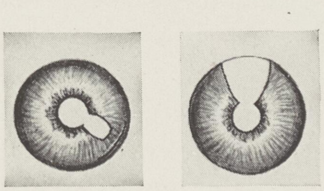


Fig. 173.—Broad Iridectomy. (After Meller.)

The iris is drawn out of the scleral wound and cut off. Since natural filtration takes place in the periphery of the angle between the iris and cornea, and since it is this angle that is obliterated in glaucoma, it becomes necessary to remove a section of iris in such a manner that no tissue will remain to obstruct filtration at that point; the iris must be detached from its root. There are two methods of doing this.

The usual method is to apply the blades of the iris scissors flat against the globe and parallel with the line of the incision. One blade lies on the scleral side of the incision and the other on the corneal side (Fig. 173). The iris is cut off with one snip of the scissors. This makes a wide coloboma (Fig. 174). The other method is employed by many English surgeons. The iris is snipped radially on each side of the iris forceps; the section is then detached from its root by traction.



A B
Fig. 174.

Unless it is the deliberate intention of the operator, no iris tissue is allowed to remain in the wound. The repositor or spatula is swept between the lips of the wound from the temporal extremity of the incision to the center, and then from the nasal extremity to the center. The instrument need not enter the anterior chamber. But where the cut edges of the iris are rolled up, it may be necessary to level them. Injury to the lens must be avoided.

Where a conjunctival flap has been formed, it must be smoothly adjusted before the eye is dressed. Both eyes are bandaged. The untreated eye may be left without a bandage after twenty-four hours, but a miotic should be instilled. The treated eye should be bandaged until the anterior chamber has been restored and the external wound has healed. Miotics may or may not be

indicated; they are useless until the anterior chamber has re-formed. Preferably, atropin is used for a possible "quiet iritis."

The *results* of iridectomy are good in the congestive type; the process is usually arrested permanently. There can be no assurance of success in the noncongestive type, for the etiological factors are not removed and drainage alone may be insufficient to arrest the progress of the glaucoma. Failures, other than those which occur in simple glaucoma, are mostly due to not detaching the iris section from its root. The coloboma does not extend into the filtration angle. This is sometimes mechanically impossible because of firm synechiæ; the patient may be so restless or disobedient that good technic cannot be practiced; or the operator can be at fault.

Complications are numerous. A hemorrhage may occur when the iris section is excised. This is not the same problem as when it happens in cataract extraction, for in glaucoma the integrity of the lens must be preserved, and efforts to remove the blood are liable to injure the lens. The blood will be absorbed in time. Where the blood clots quickly and firmly, removal can be attempted by grasping the clot with forceps.

Retinal or choroidal hemorrhage is prone to follow sudden evacuation of the anterior chamber at the time of operation. When it is delayed some days, it will be indicated by a rise in tension. It may be visible with the ophthalmoscope, but when the hemorrhage invades the vitreous there will be no fundus reflex.

The anterior chamber usually re-forms very slowly; the iris lies against the cornea. A traumatic cataract forms because of some injury to the lens during or following the operation. When the lens swells markedly the tension will likely go very high, and the cataract may have to be removed. The iris can be torn loose from its root at some point not included in the excised portion; this is iridodialysis.

A cystoid cicatrix forms when a tag or shred of iris is caught in the scleral wound. The sclera cannot heal at this point, and the aqueous will filter out along the iris tag. This is desirable from the standpoint of drainage, but undesirable from the facility with which intra-ocular infection can occur. It is recognized by a bulging of the wound.

A rare complication occurs when the vitreous gains access to the anterior chamber by way of the coloboma. This not only defeats the purpose of the operation, but it is a cause of secondary glaucoma, as has been demonstrated following cataract extraction.

An *optical iridectomy* is made with a different technic. The keratome incision is made in clear cornea; a notch is cut out of the iris next the pupil (see Fig. 174-A). As a rule this is made on the nasal side, and slightly below

the horizontal meridian of the iris. Yet it must be placed behind the clearest part of the cornea.

CORNEOSCLERAL TREPHINING

The instruments needed are an eye speculum, fixation forceps, iris scissors, 1.5 or 2 millimeter trephine (Figs. 175 and 176), Jobson's keratome knife (Fig. 177), slender forceps, and needle holder (Fig. 178). The choice of location for the operation is above or at twelve o'clock, but where there is a probability of cataract extraction later on, six o'clock may be preferable. The preparation of the patient and the positions of the surgeon and assistant are the same as in performing an iridectomy.

Fig. 175.—Elliot's Trephine.

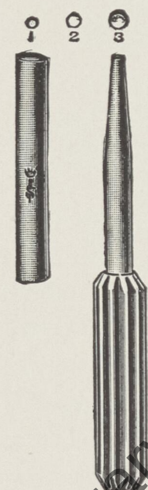


Fig. 176.—Stephenson's Trephine.

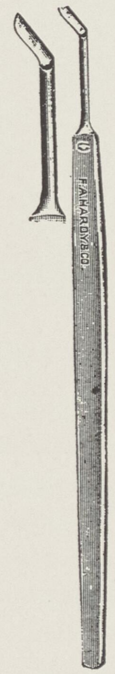


Fig. 177.

It is important to be familiar with the anatomy. The corneal epithelial layer, Bowman's membrane and a few superficial layers of substantia propria are continuous with the conjunctiva and the episclera. The main or middle portion of the substantia propria is continuous with the sclera. The deepest layers of the substantia propria, Descemet's membrane and the endothelial layer of cells are continuous with the iris and ciliary body.

Bordering the cornea the episclera is rather firmly adherent to the sclera for a distance of 3 to 5 millimeters, and the conjunctiva is very firmly adherent to the episclera for a distance of 2 or 3 millimeters. The conjunctival epithelium normally forms an elevation or ridge over the limbus (see Plate I). The success of the operation depends upon dissecting the conjunctiva and episclera from the sclera as far as the limbus, and the dissection must be done in a manner that will insure filtration. Where connective tissue is allowed to proliferate too extensively in the healing process, artificial filtration will cease.

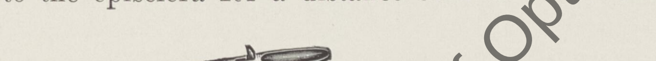


Fig. 178.

It is necessary to excise some corneal tissue, for unless a section of Descemet's membrane is removed, the endothelium will proliferate over the

opening and seal it against the outflow of aqueous. As long as aqueous has access to the lamellæ of the substantia propria, connective tissue does not proliferate, Descemet's membrane cannot be restored, and there is nothing upon which the endothelium can grow. The epithelial ridge at the limbus is a guide in the dissection.

The surgeon separates the lids with the speculum, and then takes the fixation forceps in his left hand and the scissors in his right. The patient is directed to look down as far as he can. A vertical fold of conjunctiva is grasped with the forceps as high up on the globe as possible, 9 or 10 millimeters. The conjunctiva is divided horizontally above the forceps.

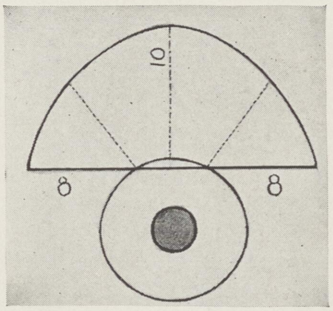


Fig. 179.
(After Elliot.)

The conjunctival incision is then enlarged to the right and to the left with the scissors so as to make a flap whose margin is roughly concentric with the limbus (Fig. 179). These cuts should come down on each side to a line drawn horizontally along the upper border of the cornea. The end of each incision should be about 8 millimeters distant from the cornea. This entire incision can be made with the single grasp of the forceps and three cuts with the scissors.

While still holding the forceps, the conjunctival flap is dissected toward the cornea until within 4 or 5 millimeters of it. The episclera is then incised horizontally, and concentrically with the conjunctival incision. With the closed points of the scissors or Jobson's special knife the dissection proceeds along the surface of the sclera to the limbus. As the dissection approaches the cornea, the width is narrowed until it is about 8 millimeters broad. The epithelial ridge must now be taken into account, for at this point the liability to buttonhole the conjunctiva is met.

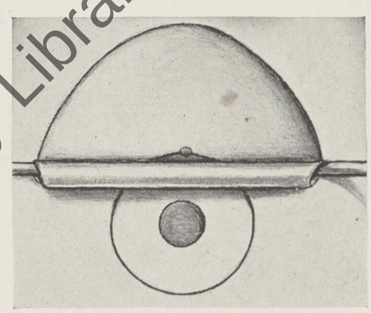


Fig. 180.

The assistant now pushes the flap down over the cornea with the closed jaws of the fixation forceps, or with a cotton-wound probe or toothpick well moistened. It may be possible to wind the flap around the probe and roll it down (Fig. 180), which is an advantage in steadying the globe, although *no pressure* should be made against it. By scraping the scleral surface with the closed blades of the scissors or with the knife, tags and shreds of tissue are removed and the surface is left clean.

With either of the same instruments the cornea is split by short side-to-side

motions. Care must be exercised to avoid either making a buttonhole or entering the anterior chamber. An effort must be made to confine the splitting process to one level of corneal lamella. Frequent inspections of progress must be made. The split cornea can usually be recognized as a steel-blue crescent, the convexity of which is directed toward the sclera.

This dissection should extend into the cornea from 1 to 2 millimeters. The surface should be cleared of any roughness. The cornea that has been subjected to congestion for some time will be very difficult to split accurately, especially as the ridge of epithelium may be absent or abnormal in appearance. From the time the speculum is inserted until the operation is completed, the cornea should be frequently moistened with warm sterile normal salt solution to prevent drying of the epithelium.

The surgeon takes the trephine in his right hand while the assistant manages the flap and steadies the eyeball. When the surgeon desires, he may steady the globe with fixation forceps applied at six o'clock, as he does while making the incision for an iridectomy. The trephine must be very sharp. Its cutting edge is placed on the sclera and slid down against the rolled flap so that at least half the drill opening will be in the cornea. The instrument must not cut into or buttonhole the flap; it is directed toward the center of the globe.

Having placed the trephine in position, the handle is rotated around its axis until a groove is cut in the tissues. The instrument must not be allowed to slide around. The cutting is occasionally interrupted to observe progress. The cornea must be divided before the sclera in order to leave a hinge on the scleral side.

The instrument must not enter the eyeball on account of the almost certain damage that would be done. The lightest effective pressure must be used. Elliot's trephine was designed to carry in the handle the exact weight needed. Stephenson's trephine is tapered and Brown Pusey's is shouldered to guard against boring too deeply.

When the disk has been cut, the surgeon feels a sensation of suction or nonresistance on the instrument; the patient feels a sudden slight pain, and aqueous escapes around the instrument. The instrument is removed and the iris bulges into the wound so that the pupil becomes pear shaped. With a hazy cornea this may escape notice.

Usually the iris pushes the disk flap outwardly, although the scleral hinge holds it on that side. When the iris does not present in the opening and the aqueous is trickling out, the conjunctival flap is replaced at once. When the iris is bound to the cornea it will not appear in the wound, and aqueous will not flow. Such an iris is atrophic and brittle. An effort may be made to pull it into the wound and excise a portion of it, or an opening may be made in it

with the very point of a sharp knife. When aqueous flows, the trephine probably made an opening in it; this would communicate with the posterior chamber.

When the iris presents, it and the disk flap are seized with forceps and both are cut off with a single snip of the scissors. This should be done at once, for when the pressure in the anterior chamber subsides, the iris resumes its natural position. It is almost certain to obstruct the opening later. Having once settled back it must be fished for with slender forceps.

Some operators prefer a complete iridectomy, and draw the iris out of the wound so that they can divide the pupillary border. Others make a peripheral iridectomy when they cut off the trephine button. The latter leaves an intact pupillary border, and miotics have a more positive effect when the circular muscle-fibers surrounding the pupil have not been divided. The complete iridectomy has been advocated in the congestive type, and the peripheral in the noncongestive type of glaucoma.

When any iris tissue remains in the trephine hole it should be washed out with a fine stream of sterile normal salt solution. The episcleral and conjunctival flap is smoothed out carefully and replaced. The cut edges of the conjunctiva are united by two silk sutures. The conjunctival sac is cleansed with an aseptic solution. The lids are released from the speculum, the upper lid is lifted, the patient is directed to look up, and the lid is lowered on to the globe. Both eyes are bandaged.

The eye is inspected after twenty-four hours. Very gentle massage may be given to encourage filtration. Atropin is instilled unless the pupil is well dilated, because of the liability of "quiet iritis." When a miotic is used at this stage it encourages the formation of posterior synechiæ in the congestive type. The operated eye only is bandaged, and the patient is allowed to sit up in bed. After healing, treatment is continued along the lines mentioned under local management.

Complications arise from intra-ocular hemorrhage, detached choroid, loss of vitreous, forward displacement of the lens against the trephine hole, obstruction by scar tissue, iritis, and late infection.

The manner of forming and dissecting the conjunctival flap confers certain advantages. The conjunctiva not only reunites very firmly along the line of incision, but it forms adhesions with the episclera beneath that line. If the incision met the limbus on each side of the trephine opening, scar tissue would arrest filtration. The gap between either extremity of the incision and the limbus practically insures a scar-free exit both temporally and nasally.

Should an impermeable scar form along the line of the incision for the episcleral flap, a bleb will form but no filtration will take place and the tension will rise. After proper sterilization of the operative field, a Graefe cataract

knife is thrust under the conjunctiva toward the bleb. The puncture in the conjunctiva should be at least 6 millimeters distant from the bleb. The flow of aqueous is reëstablished by dividing the episcleral scar subconjunctivally with the point of the knife.

MISCELLANEOUS OPERATIONS

Although the operations of iridectomy and corneoscleral trephining are almost universally practiced, a knowledge of the essential features of other operative procedures is advisable. Either of the two most practiced operations may fail to lower the tension or arrest the progress of blindness. The eye will have to be operated on again and again perhaps, or a different operation may be desired for the same or for the second eye. The surgeon should be at liberty to combine the good points of two or more operations.

Reese modified the technic for iridectomy and added features from other operations. The conjunctiva is grasped in such a manner as to include the tendon of the inferior rectus. With the forceps the operator rotates the cornea downward and steadies the globe. A keratome of 21 degree angle is used. The point enters the conjunctiva 7 millimeters from the limbus, and is pushed subconjunctivally to within 2 millimeters of the margin of the cornea. The handle of the instrument is raised so that the blade will enter the anterior chamber by making an oblique cut in the sclera. The cut is made mostly with the entire length of the right edge of the blade. It is designed to have the incision 8 millimeters in length, and 2 millimeters distant from the limbus at all points (see Fig. 172).

The instrument is smoothly withdrawn in the same plane by which it entered; there should be no loss of aqueous. The conjunctiva is replaced. From each extremity of the conjunctival incision a cut is made upward and backward. The assistant now takes the fixation forceps.

The iridectomy is made; 1 millimeter of iris is grasped as near the pupillary border as possible, and as it is pulled out it is everted so that the posterior surface is anterior. With curved scissors, the concavity of whose blades is turned toward the right, a snip is made in the iris to the right of the iris forceps. This should extend to the ciliary attachment. The iris is then cut or torn across at its root beneath the forceps, and the section is detached by cutting on the left side of the forceps.

The conjunctival flap is pulled forward to expose the anterior lip of the scleral wound, and several nicks are made in that lip with a scleral punch. The flap is then replaced and the eye is dressed. Gentle massage is begun on the following day, and is continued as long as the tension justifies it.

Lagrange devised a *sclerectomy* with or without iridectomy. With a Graefe knife a puncture and counterpuncture are made 1 millimeter behind the limbus and 7 millimeters apart. The plane of the blade is turned to bevel the sclera upward and backward (Fig. 181). The incision is carried subconjunctivally so as to form a flap of that membrane 5 or 6 millimeters long. This flap is drawn forward over the cornea. When the anterior lip of the scleral wound comes into view a piece of it is excised with small sharply curved scissors (Fig. 182). An iridectomy is made or omitted as desired, as described on page 253 (see Figs. 173 and 174), and the flap is replaced.

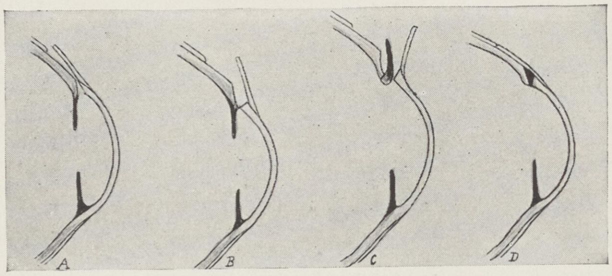


Fig. 181.—Lagrange's Sclerectomy.

Holth planned a dissection similar to that used for trephining, except that the cornea is not split. A small opening is made into the anterior chamber, and a hole is made in the sclera next the cornea with a special punch forceps.

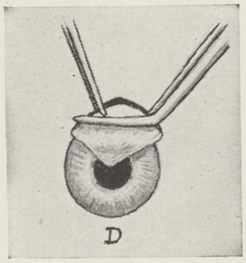


Fig. 182.
(After Meller.)

Anterior sclerotomy is performed with a Graefe knife by making a puncture and counterpuncture 1 millimeter behind the limbus, and 2 millimeters below and parallel with a horizontal line drawn across the upper border of the cornea. The knife divides the sclera, but leaves an intact conjunctival bridge above (Fig. 183).

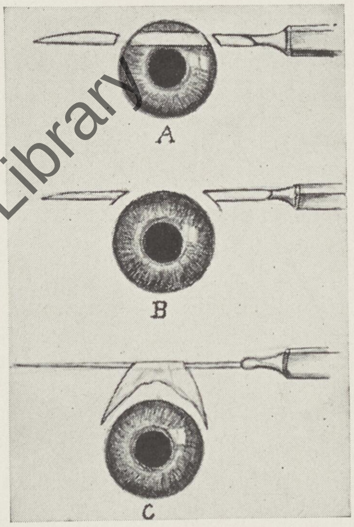


Fig. 183.
(After Meller.)

Posterior sclerotomy is performed with a Graefe knife by entering the globe 10 millimeters behind the limbus between the lateral and inferior recti muscles. The plane of the blade is meridional or anteroposterior. The point of the knife is directed toward the center of the eyeball, and it should penetrate the coats for 4 or 5 millimeters. As the blade is withdrawn it is turned at a right angle to enlarge the opening (Fig. 184). This operation is often used a half hour or so before performing one of the anterior operations in hemorrhagic glaucoma. It lowers the tension and deepens the anterior chamber temporarily.

Iridotasis means incarcerating a segment of iris in a scleral wound. Wilder makes a conjunctival flap like that used for trephining, but not so broad. The middle part is dissected down to expose 4 or 5 millimeters of the limbus. A keratome incision 4 millimeters long is made into the anterior chamber from a point 1 millimeter behind the margin of the cornea. With delicate iris forceps the iris is grasped at its pupillary border and pulled into the wound, so that the posterior or retinal surface is anterior.

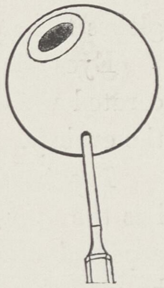


Fig. 184.

The intention is to incarcerate the sphincter of the iris in the wound in this position, for the retinal surface will not unite to cornea or sclera. As long as no healing occurs, the aqueous will filter out along the fistulous tract. The conjunctival flap is replaced and that eye only is bandaged. Atropin is instilled on the following day. This operation is not indicated where there is a likelihood of having to remove a cataract from that eye.

Cyclodialysis or Heine's operation seeks to establish posterior drainage. A conjunctival flap is formed between the inferior and lateral recti muscles with its base toward the limbus. Five millimeters behind the corneal margin a short equatorial incision is made through the sclera only. A thin spatula is introduced into the wound and by rocking motions is passed forward flatly between the ciliary body and the sclera into the anterior chamber (Fig. 185). The spatula is then moved from side to side until about a third of the circumference of the ciliary body has been detached from the sclera. If the spatula is slowly withdrawn, there should be little or no loss of aqueous. The reduction of tension is gradual.

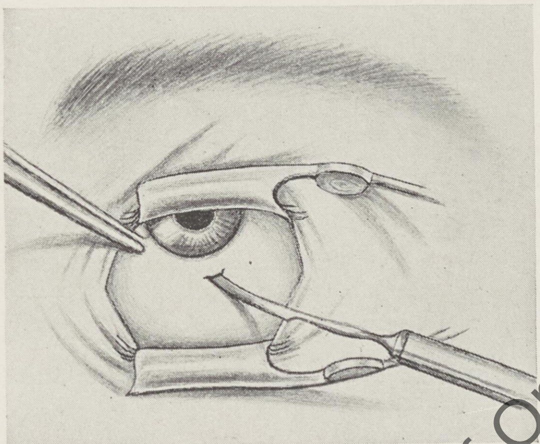


Fig. 185.—Heine's Cyclodialysis.

Iridencleisis as devised by Holth requires a complete iridectomy. One of the iris pillars is drawn into the wound and becomes incarcerated. Filtration takes place alongside this tissue.

The criticism of the methods which leave iris tissue in the scleral incision is based on the ease with which late infection or even sympathetic ophthalmia can occur. The contents of the globe are protected by the conjunctiva alone.

Herbert makes a scleral flap and leaves it to act as a valve.

The tension can be reduced, at least temporarily, by subconjunctival punc-

tures of the limbus. A narrow Graefe knife is thrust beneath the conjunctiva 5 or 6 millimeters from the limbus; with the point of the knife several punctures are made in the angle of the anterior chamber, each with the blade in a different plane.

It is permissible to operate on both eyes at once in glaucoma, but bilateral operations are not generally approved in other conditions which require the globes to be opened.

Eyes that are hopelessly blind from glaucoma must be enucleated or eviscerated when the pain cannot be controlled by any of the operations that save the eyeball.

Treatment of buphthalmos has been summed up by Patton. Trephining has the approval of a majority of operators, but it should be done early and repeated where the tension is not reduced or when it returns. Miotics should be used for at least a year after operation. The tension can be temporarily reduced by subconjunctival punctures of the limbus. The operative prognosis is guarded. Benefits are derived from about half of the operations performed on buphthalmic eyes.

The treatment of iris bombé is shown in Figures 137 and 138.

HYPOTONY

Diminished intra-ocular pressure occurs when the aqueous flows off more rapidly than it forms. This can come from diminished secretion or from accelerated filtration.

The secretion is diminished in contusions, injury to the vitreous, some cases of intra-ocular hemorrhage, rupture of the choroid, detachment of the retina, paralysis of the sympathetic nerve, herpes zoster ophthalmicus, ophthalmomalacia, iridocyclitis, exudative choroiditis, interstitial keratitis, phthisis bulbi, pressure on the ciliary body by a dislocated lens, cyclodialysis, and in fluid vitreous. When softening of the eyeball occurs in diabetic coma the prognosis is grave. The secretion can be temporarily diminished by a pressure bandage that is too tight.

The aqueous escapes too rapidly to maintain normal intra-ocular pressure when a corneal ulcer perforates, or while a penetrating wound remains open. Operations for glaucoma may reduce the tension below normal. Temporary hypotension sometimes follows the use of miotics and massage.

The effects of continued hypotony are wrinkling of Bowman's or Descemet's membranes, which may be recognized as fine lines or checks in the cornea. Myopia may be noted. The eyeball may shrink. The treatment depends upon the cause.

CHAPTER XV

THE CRYSTALLINE LENS

ESSENTIALS OF THE EMBRYOLOGY AND ANATOMY

The crystalline lens is a clear transparent body of biconvex shape suspended between the pupil and the vitreous. It is 9 millimeters in diameter and 3.6 millimeters thick while at rest, and 4.0 millimeters thick while in a state of maximum accommodation. It has an anterior and a posterior surface, and an anterior and a posterior pole. An imaginary line connecting the poles is the axis. The circumference of the lens is called the equator.

Embryologically, the lens is formed from a bit of specialized ectoderm which is called the "anlage" of the lens. When the anlage is deposited on an ectodermal surface it induces localized invagination of that surface, which when closed and pinched off from surrounding ectoderm becomes the lens vesicle. By the process of invagination the basal membrane becomes the outermost layer of the vesicle.

As the cells of the basal membrane of epidermis grow they force older cells toward the surface, where they become devitalized and are cast off. As the cells of the basal membrane of the lens grow they crowd older cells toward the center where they become devitalized, but cannot be cast off, absorbed, or carried away. The devitalized cells lose their nuclei, and collectively they form the nucleus of the lens body.

The basal membrane of the lens consists of a single layer of cells which covers the anterior surface and overlaps the equator. The cells on the posterior surface are expended during fetal life. The cells and their processes are hexagonal in shape, which insures the maximum number in the smallest space. The processes are called fibers, and they are held together by a cement substance.

The growth of the fibers is from the anterior surface, over the equator, to the posterior surface. Thus the lens is built up of concentric laminæ of fibers. As the fibers which grow from one group of cells meet with fibers from other groups of cells, sutures or septa are formed in the substance of the lens. These suture lines are recognized in youth and early adult life as "Y" figures; the anterior one is upright (Fig. 186), and the posterior is inverted. As age advances and the fibers accumulate, the sutures increase in number.

The lens continues to increase in size throughout life by the growth of new fibers. The nucleus is formed of shrunken, hardened or sclerosed dead fibers; it grows in size by accrementition. The two zones of living and dead fibers are fairly well defined. The living fibers constitute a colorless mass, called the cortex, which entirely envelops the nucleus. The nucleus is practically colorless in early life, but as it increases in size and density it acquires a yellowish tint. The cortex is comparatively soft, and the nucleus is hard.

The lens nucleus consists of three parts or layers which are called the fetal, the adult and the senile nucleus respectively. So long as the nucleus is free of color it reflects no light and the pupil looks black. In age, after the nucleus has acquired color, density and flatness, it reflects enough light to give a "gray reflex" or a gray pupil. This gray or senile reflex is often diagnosed as cataract by the uninitiated.

The lens is enveloped by a structureless or hyaline capsule that prevents aqueous from gaining access to the lens fibers; the latter are highly hygroscopic. A knowledge of this protective device enables the surgeon to "ripen" a cataract by the operation of "discission."

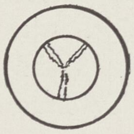


Fig. 186.

The lens rests in the patellar fossa of the vitreous body posteriorly, while anteriorly it supports the pupillary border of the iris. Its equatorial border lies within the circle of the tips of the ciliary processes, and it is separated from them by a space of 0.5 to 1 millimeter. It is held in position by the suspensory ligament of the lens or the zonule of Zinn, and by adhesion to the anterior border layer of the vitreous in the deepest part of the patellar fossa.

The zonule or zonula is formed of fine fibers which arise from the ciliary body. The fibers that arise from near the ora serrata course forward along the inner surface of the ciliary body and are inserted into the anterior capsule of the lens. Fibers from the anterior portion of the ciliary body are inserted into the anterior capsule, but chiefly into the posterior capsule. Short fibers from between the ciliary processes are inserted into the margin of the lens.

The arrangement of fibers as they cross the equator of the lens from behind forward and from before backward forms the circumlental space or the canal of Petit, which completely surrounds the lens. The aqueous filters through the spaces between the zonular fibers to both the vitreous and the posterior aqueous chambers.

The lens, until about eight weeks before birth, is supplied by the anterior and the posterior tunica vasculosis lentis. The anterior tunic is derived from the lesser vascular circle of the iris, and remnants of it, which may amount to a persistent pupillary membrane, are always attached to the anterior surface of the iris outside of the sphincter zone over the site of the lesser circle (see

Fig. 142). They may adhere to the anterior lens capsule and account for minute opacities, but they do not prevent dilatation of the pupil.

Posterior synechiæ arise from the pupillary margin of the iris, and interfere with the dilatation of the pupil. When detached or torn loose they leave brown spots on the lens.

The posterior tunic is made up of branches derived from the hyaloid artery. Remnants of it often remain on the posterior lens capsule. A common one is called a Mittendorf's dot; it is found slightly medial to and below the posterior pole where the hyaloid artery had contact with the lens. It has no pathological significance. These congenital remnants are usually bilateral; they are occasionally associated with other anomalies.

The lens is without a blood supply after birth. It is thought that the aqueous supplies nourishment by diffusion or osmosis; there are no channels for circulating fluids. When the constitution of the aqueous is changed pathologically the lens may be damaged. The lens has no known nerve supply. It is subject to degenerative changes but not to inflammation.

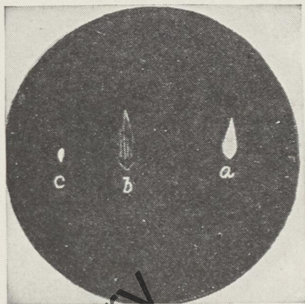


Fig. 187.—Purkinje-Sanson Images.

The *function* of the lens is to focus rays of light from an object so that they will form an image of the object on the retina. The lens is nearly spherical in early fetal life. It flattens as the eyeball grows, but retains elasticity, and in a state of accommodation it resumes something of its original spherical form. Anteroposterior thickening of the lens is essential to accommodation. As the nucleus increases in size and rigidity the elasticity diminishes. When a loss of elasticity must be compensated by the use of lenses for reading or near work, the age of presbyopia has arrived.

The *examination* of the lens is best undertaken in a dark room with the pupil dilated. The presence of the lens is demonstrated by Purkinje-Sanson's images. A lighted candle is held on a level with the eye and to one side, while the examiner looks into the eye from the opposite side. A large upright image of the candle flame is mirrored from the anterior surface of the cornea. A small inverted image is reflected from the posterior surface of the lens; it is about 7 millimeters behind the corneal image. A very large upright image may be dimly seen on the anterior surface of the lens (Fig. 187).

Normally, the pupillary border of the iris is supported by the lens. When the center of the anterior chamber is shallow, the lens may be swollen or it may be pushed forward by something behind it. When the anterior chamber is deep and the iris trembles, iridodonesis, with movements of the eyes or head,

the lens is shrunken, dislocated or absent, or the vitreous does not support it.

With oblique illumination the iris casts a shadow on the cataractous lens. The pupil will be uniformly black unless there is a sclerosis of the lens; then the gray or senile reflex will be seen (Fig. 188). When opacities are present they will reflect any light that meets them, and appear in the field as gray or white dots, lines or figures. In ripe or mature cataract the pupil will be uniformly white; the iris will cast no shadow upon the lens that is totally opaque. The breadth of the iris shadow, which is crescentic on the side from which the light comes (Fig. 189), depends upon the thickness of the opacity. This test is used to ascertain the presence of a cataract and its degree of development.

Fig. 188.

Small opacities may be in the lens, in the aqueous or in the cornea (see Figs. 93 and 94). If the examiner will "sight" the opacity with reference to the margin of the pupil, and then move his head so as to bring both the opacity and the pupil margin into his line of sight at one time, he may estimate the location of the opacity. If, while he sights the pupil border, the opacity moves in the direction his head moves, it is behind the pupil; if opposite, it is in front of the pupil.

If, while he sights the opacity, the pupil border moves in the same direction that his head moves, the opacity is in front of the pupil; when it does not move, the opacity is in the plane of the pupil; and when the pupil border moves in the opposite direction to his head movement, the opacity is behind the pupil (see Fig. 95). The more rapidly the pupil border moves, the farther the opacity is from the plane of the pupil. A reasonable degree of accuracy accrues from practice.

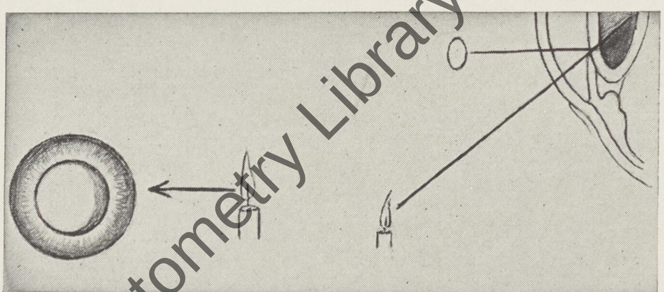


Fig. 189.—Iris Shadows on Lens. *O*, Observer. (After Fuchs.)

The objective examination is completed with the ophthalmoscope. The normal fundus gives a clear red reflex when illuminated if the media are transparent. This reflex is due to light coming from the fundus, therefore the rays are direct. Opacities in the lens obstruct the rays and stand out as dark spots or lines in a red field. When a lens is totally opaque, the pupil is gray or white and there is no fundus reflex.

To detect lens opacities, the observer looks at the pupil through a plus 7 lens in the sight hole of the ophthalmoscope; his distance from the observed eye is about 15 centimeters (6 inches). The examiner varies the distance un-

til the opacities are in focus; then he moves his head from side to side and in this way estimates the area and situation of the opacities.

The subjective examination consists of a visual and a field test. The best vision is ascertained with trial case lenses. The field of vision is found with a perimeter or a campimeter. When the lens is totally opaque, the patient has only light perception, l.p. He should also have light projection. This is found by covering the fellow eye with a card, and holding a light in various positions before the eye being tested. The patient has light projection when he can point to the light in these several positions.

CATARACT

Varieties, Classification, and Etiology

The commonest pathological change that occurs in the lens is cataract. This includes any form of lenticular opacification. The opacities vary in their nature, extent and location. Kirkpatrick (*Cataract and Its Treatment*) classified them as follows: The partial and nonprogressive group comprises anterior and posterior capsular or polar, central, fusiform, coralliform, disk-shaped, Doyne's discoid, punctate, lamellar or perinuclear or zonular, and anterior and posterior cortical; the progressive group comprises presenile, senile, glassblowers' and traumatic. Where the lens is very dark it is called "black cataract." The members of the progressive group pass through incipient, immature, mature and hypermature stages.

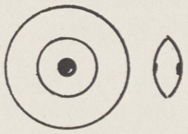


FIG. 190.

Anterior Polar cataract (Fig. 190) is an intensely white, sharply circumscribed spot in the center of the anterior portion of the lens. Sometimes its surface is even with that of the lens, while at other times it protrudes cone shaped into the anterior chamber and is then called a pyramidal cataract (Fig. 191).

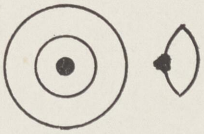


FIG. 191.

This variety is congenital or acquired. The congenital form is frequently bilateral. It is thought to be due to an intra-uterine keratitis. The lens and cornea come into contact; when separation occurs an opaque spot remains on the lens. Some irregularity in the development of the eye may be responsible for some cases. The acquired form may be due to perforating ulceration of the cornea, such as is found occasionally in ophthalmia neonatorum. The lens comes forward against the corneal aperture; when the cornea heals and the lens separates from it, the point of contact is permanently marked on the lens. Treatment is unnecessary unless the opacity interferes with vision. Discission and linear extraction are the operations usually performed for it.

Posterior Polar cataract (see Fig. 190) appears at the posterior pole of the lens, and is comparable in appearance to the anterior polar form. It is thought to be due to a remnant of the hyaloid artery, but should not be confused with a Mittendorf's dot which is always dark. It is demonstrable by direct ophthalmoscopy, but not by oblique illumination. When the opacity appears elsewhere than at the pole of the lens it is not a polar cataract. Treatment is unnecessary.

Posterior Capsular cataract can occur in any part of the posterior capsule.

Fig. 192.

Central cataract (Fig. 192) appears in the center of the lens body, and may be complicated by other opacities. It is congenital. It may be let alone, an optical iridectomy may be performed, or the lens may be removed by a linear extraction following a preliminary discission.

Fusiform, axial or spindle-shaped cataract (Fig. 193) extends from one lens pole to the other, and is a form of nuclear opacity. A *coralliform cataract* is so named from its resemblance to a coral formation. Its appearance suggests an anomalous development along the lines of the sutures.

Fig. 193.

Fig. 194.

Disk-shaped cataract (Fig. 194) is thin in the center and thicker toward the equator. A cross section resembles a dumbbell. It would seem that the nucleus had failed to develop. Its removal may be attempted by discission.

Doyne's Discoid cataract (Fig. 195) is found as a circumscribed disk between the nucleus and the posterior pole. It has been placed in the hereditary class. It is congenital, bilateral, symmetrical and stationary. It may obstruct a view of the fundus, or it may not materially interfere with vision.

Fig. 195.—White Opacity in Black Pupil.

Punctate cataract shows many small dots, and short radiating lines appear in the cortex just inside the equator of the lens. Since it is outside the area of the pupil, vision is not affected. It is congenital, generally bilateral and nonprogressive, but seems to predispose to senile cataract. With a loupe the delicate opacities are seen more readily, as well as a bluish color which has given rise to the name cataracta cœrulea; these can be easily extracted in their capsules without iridectomy.

Lamellar, perinuclear or zonular cataract can only be seen in its whole extent with a well dilated pupil. It is a central disk whose periphery is darker than its center. The lens nucleus is clear, but it is completely enclosed by an opaque envelope of rather uniform thickness (Fig. 196). Outside this the cortex is clear. Lens "spokes" are frequently found outside the periphery

of the opacity (Fig. 197). When closely examined these spokes are found to be continuous over the anterior and posterior surfaces of the borders of the cataract (Fig. 198); they are called "riders."

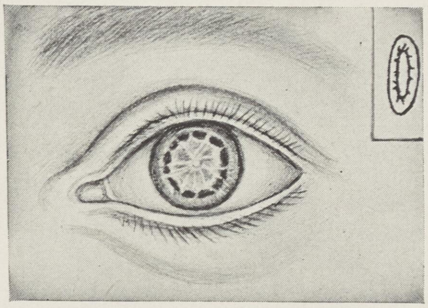


Fig. 196.—Lamellar Cataract.

In children this is the most common variety of cataract; it may be congenital or hereditary, it is usually stationary, and is generally bilateral. As it lies in the pupillary area, the impairment of vision depends entirely upon the density of the opacity. Myopia is a common complication, possibly more apparent than real; the patient holds print or objects near the eyes to enlarge the retinal images. Suitable lenses should be worn.

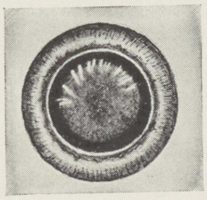


Fig. 197.

Lamellar cataract is often found in children who have been the subjects of convulsive seizures or of tetany, both of which are characterized by periods of malnutrition. The teeth are marked by horizontal furrows in the enamel, and resemble the malformations that have usually been ascribed to rickets. The number of the teeth so marked and the size of the cataract give fairly reliable information as to the approximate age of the patient at the time the opacity began.

Fig. 198.

Treatment should not be instituted until it is determined that the opacity is stationary or progressive, and that vision is materially improved with a dilated pupil. Where the opacity is relatively small and stationary, and the vision is fair through the periphery of the lens, an optical iridectomy is indicated. When the opacity is relatively large, or progressive, and the vision is poor, the lens may be absorbed after repeated discissions, or it may be extracted through a linear incision. The latter procedures necessitate the constant wearing of lenses for both distant and near vision; accommodation is lost.

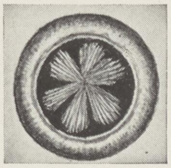


Fig. 199.

Anterior and Posterior Cortical cataracts are formed of lines which radiate from one lens pole or the other toward the equator; the figure is that of a star or a rosette (Fig. 199). Since this opacity occasionally accompanies such diseases as retinitis pigmentosa and choroiditis, treatment of the cataract for the improvement of vision will likely prove temporary and disappointing. It is usually stationary, but may become progressive. It has followed concussion injuries of the eye.

Glassblowers' cataract occurs among workers who are exposed to intense heat and glare. It is bilateral, and begins in the posterior layers of the cortex as a posterior cortical cataract. The greatest density lies at the pole of the lens, but this fades gradually toward the equator. It is progressive. In early cases the opacity has a brassy saucer-shaped appearance by oblique illumination. When the cataract is mature it is pearly. It is treated by extraction. The left eye is said to be the more frequently affected.

Traumatic cataract is caused by a variety of violent means, all of which injure the epithelium and allow aqueous to gain access to the lens fibers. Concussions or penetrating wounds are usually responsible.

When due to concussion the cataract belongs to the posterior cortical variety; it may remain stationary or disappear. The anterior lens epithelium is sometimes disturbed by lightning or electric shocks, with resulting opacity. The aqueous enters the substance of the lens through the disordered epithelium. In accidents which merely injure the epithelium, the aqueous cannot enter the cortex until the capsule becomes permeable to it. The development of the opacity is delayed. Where the force ruptures or tears the capsule the permeation begins at once and opacification is rapid.

Penetrating injuries expose the cortical fibers to aqueous. They absorb water, swell, become opaque and finally undergo degeneration and absorption (Fig. 200). A hard nucleus does not absorb water, and it is not affected.

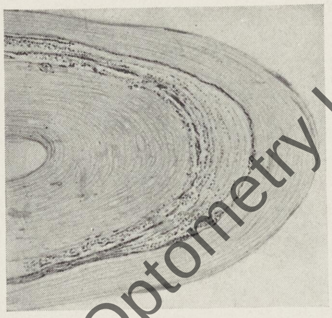


Fig. 200.—Lens Degeneration.
(After Fuchs.)

Young persons, who have a soft nucleus, may have useful vision with a compensating spectacle lens. In older persons the nucleus will have to be extracted.

The lens fibers swell very rapidly in some instances and produce an *intumescent cataract*. A secondary glaucoma may follow. A paracentesis will often lower the tension sufficiently to permit an operation on the lens. It may be removed by a linear extraction; a preliminary iridectomy, followed by a flap extraction, will be indicated in other cases.

Presenile or juvenile cataract can occur at any age. Congenital anomalies may be present. The subject may have diabetes. When the cortex liquefies, the nucleus is free to move about in the capsule; this constitutes a *morgagnian* cataract (Fig. 201). These can be treated by discission, or by linear or flap extraction.

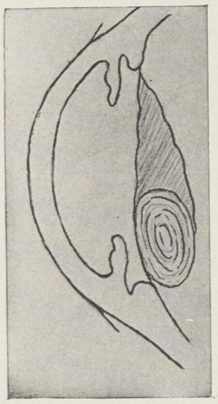


Fig. 201.—Morgagnian Cataract.

Secondary or complicated cataracts follow some intraocular diseases such as iritis, iridocyclitis, glaucoma, detachment of the retina, hemorrhages (see Plate IV, Fig. F), and perforating ulcers of the cornea.

Etiology of cataract is largely speculative. Of many individuals of the same age living under similar conditions, very few develop cataract. For every cause assigned, more persons escape than are afflicted. Statistics prove that there is a strong hereditary influence, in which the element of anticipation occasionally occurs. Congenital cataracts are ascribed to intra-uterine eye disease or to faulty development of the lens; they are frequently bilateral, often seem to be inherited, and may be either partial or nonprogressive in type.

Certain diseases, probably by disturbing the nutrition of the lens, seem to predispose to cataract. Diabetic cataract is not uncommon, especially when the cataract develops rapidly; when it develops slowly in a diabetic patient, it is more probably a senile cataract. Toxemias are sometimes followed by lenticular opacities. Tetanus has been followed by lamellar cataract, especially in children. Certain poisons as naphthalin and ergot have seemed to produce cataract; they probably induce nutritive changes. Eye-strain, hyperopia and hyperopic astigmatism predispose to cataract, but these factors are themselves frequently hereditary.

Vossius Ring cataract is thought to be due to an impact of the pupillary border of the iris against the lens capsule, as it is found in a location corresponding to that border. It gradually disappears.

Senile cataract, so called, is the most important variety because of the large number of cases that come under that classification. Disturbances of the endocrine balance, as observed in diabetes and some of the toxemias, offer some reflections on the possible causation of senile cataract. Senility is marked by a functional failure of the glands associated with the faculty of generation. Where such a failure is balanced in the activities of other glands, the individual will probably continue in good health. When compensation fails, the individual becomes subject to chronic infections, faulty food assimilation and disturbances in metabolism.

It is significant that a thyroidectomy is occasionally followed by the de-

velopment of a cataract. This may be due to a loss of the thyroid secretions or to an injury to the parathyroids. Nature makes an effort to provide for the aged, for comparatively few old people are afflicted with blindness from cataract. The element of conservation appears to be emphasized in that while senile cataract is generally bilateral, one lens usually withstands the process of opacification longer than the other.

Symptoms, Signs of Cataract

Symptoms of cataract of the progressive type begin with a mistiness of vision that deepens into fogginess and finally leads to a blurring of objects. This impairment may be diffuse or it may appear as a small circumscribed cloud in the line of direct vision. When the opacity is central, vision will be improved in diminished light because the pupil is larger than it is in bright light; a mydriatic will artificially produce a similar improvement. When the opacity is peripheral, vision is better in bright light with a contracted pupil. The visual acuity cannot be estimated without the use of the best lenses for that patient. The patient frequently complains of floating bodies in the field of vision.

Changes in the refraction are due to the changes that take place in the lens. Astigmatism may appear in either of two ways: (1) Corneal astigmatism may be compensated by an action of the ciliary muscle which alters the shape of the lens. When the lens becomes too rigid to respond to the action of the muscle, the corneal astigmatism cannot be compensated. (2) The hardening process may occur without uniformity; this constitutes lenticular astigmatism. If the lenticular astigmatism is irregular, the patient may be annoyed by multiple images.

Through the increased density of the fibers, or the increase in the curvature of the surfaces, the lens may become more highly refractive; these changes produce myopia. The process may go on until the near-point of the eye is at the reading distance, and the patient has "second sight"; the distance vision will be myopic.

When the lens flattens, the eye becomes hyperopic or the existing hyperopia increases. Increased hyperopia may depend upon an altered state of the nutrition of the lens which decreases its refractive power.

When pain and photophobia are present they are usually explained as being due to asthenopia. Sometimes halos are seen around artificial lights; it is claimed that this occurs in cataract cases only when the lights are at a certain distance from that particular patient.

The opacification proceeds until the patient is unable to distinguish ob-

jects, but retains perception for light unless there is some pathological condition behind the lens.

The patient who is blind solely from cataract should have good light perception and light projection. He should be able to recognize colors when colored glass or test lenses are held between his eye and a light; this is color perception. The pupil should contract to light, and also to convergence when the patient is directed to look at his own hand held before his face. In these tests the fellow eye should be excluded by a pad.

Signs of cataract are found by the use of the condensing lens, the loupe and the ophthalmoscope. The pupil must be dilated, and two drops of 2 per cent solution of homatropin will usually suffice for this examination, which should be conducted in a darkened room. Light is focused on the pupil with the condensing lens. When the entire lens is opaque the iris will cast no shadow upon it. When the nucleus is opaque the iris will cast a crescentic shadow upon the lens. The width of the shadow will afford an estimate of the depth of the opacity behind the border of the pupil (Fig. 202).

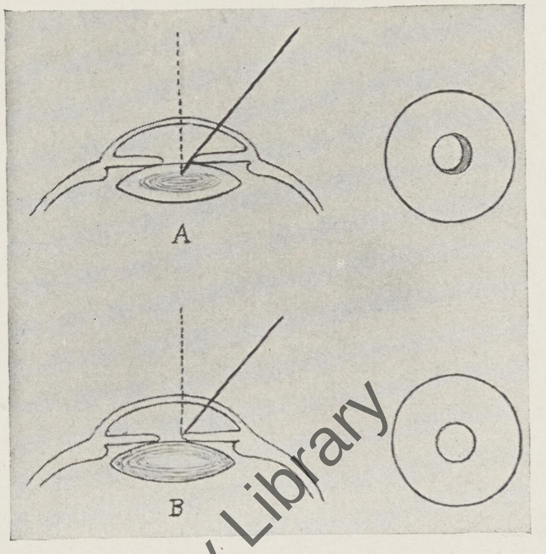


Fig. 202.—*A*, Iris Shadow on Opaque Nucleus; *B*, No Shadow on Opaque Cortex. (After Parsons.)

With the loupe and the condensing lens the pupil may be searched for opaque dots, lines or figures which will reflect light and therefore appear white or gray. Lenses in aged persons often reflect light from the whole width of the pupil; where the fundus can be seen clearly through all parts of a dilated pupil with the ophthalmoscope, senile cataract does not exist. Attention to this method of distinction will save confusion in pronouncing a lens cataractous when the patient has simple or noncongestive glaucoma.

When light is thrown into the eye from a distance with the ophthalmoscope or a head mirror, the red fundus reflex should be seen. This is absent in mature cataract. Dots, lines or figures that are seen against this background are black. By the parallactic test, opacities of the cornea and vitreous may be excluded.

Progressive cataract, or so-called senile cataract, when undisturbed, passes through three stages into a fourth. They are: Incipient cataract (early),

intumescent cataract (swelling), mature (ripe) and hypermature cataract (overripe).

Incipient cataract begins in one of three ways or in a combination of them. The commonest change is noted as triangular or wedge-shaped opacities which radiate from near the axis toward the equator like the spokes in a wheel; the bases are situated in the periphery and the points are directed toward the center. They may appear as lines. The opacities are gray to oblique illumination and black to ophthalmoscopic examination. The lens substance between them is transparent. When the nucleus is affected first, the opacity appears in the pupillary area. Sometimes the opacity is faint and general, and looks like a diffuse cloud of dust particles.

The opacities may remain stationary for a long period, and impair vision but little. The laity takes alarm at the word "cataract." The announcement fills the patient with apprehension; it is better to speak of "opacities" and then inform a relative as to the true condition.

Low myopia is a common development, and so is astigmatism. When the latter is irregular, glasses will be unsatisfactory. Yet the refraction must be kept as closely corrected as possible. Amber, blue or smoked glasses may be ordered in addition. The anterior chamber is usually of normal depth in this stage.

Intumescent cataract presents a hazy bluish color with a silky lustrous surface; the lens sutures are plainly displayed. The iris casts a definite shadow, but a similar shadow can be cast upon a hypermature cataract or on a shrunken lens. The swollen lens crowds the iris forward, and the anterior chamber is shallow. The intra-ocular pressure may be somewhat elevated in this stage.

Mature cataract exists when the lens is completely opaque, the spokes or wedge-shaped areas can no longer be detected, the iris casts no shadow, the lens sutures are still visible (see Plate IV, Fig. F), the anterior chamber is back to normal depth and the patient's vision is reduced to light perception. The lens shrinks from loss of fluid. Its surface is dull gray, light brown or yellow. Until the cataract is mature the spokes are visible; just before they disappear they become somewhat glittering when the light strikes them at certain angles (see Fig. 197).

Hypermature cataract usually follows a long period of maturity. It is formed in one of two ways; the cortex either shrinks or it breaks down. In the beginning of cataract formation the lens fibers shrink so that spaces are formed between them. These spaces are occupied by foreign fluid. The cortical fibers absorb this fluid, swell and become cloudy (see Fig. 200). When the fluid leaves the lens the cortex shrinks; where it remains a long time the cortex breaks down and liquefies. In either case the cortex is destroyed.

The lens nucleus is comparatively small up to the age of thirty-five years; after that age it increases in size continuously. The cortex becomes pulpy where the nucleus remains small, and this constitutes a "soft cataract." Where the nucleus enlarges pathologically the cortex shrinks upon it or disappears; this forms a "hard cataract." In either case the lens flattens and becomes smaller in diameter; the whole lens is diminished in size.

A soft cataract has a flat uniformly yellowish appearance without markings. A hard cataract has a flat brownish or yellowish appearance; the suture lines disappear as such, and white spots appear in the lens. The yellowish or brownish appearance of a hard cataract is due to a faint reflex from the nucleus; it is best seen by oblique illumination. An estimate of the size of the nucleus is afforded by this reflex.

When the lens shrinks the anterior chamber deepens. The iris is apt to be quite flat and may again cast a shadow on the lens; when the iris does not have the support of the lens it will tremble with movements of the head and eyes. The shrinking of the lens stretches the zonular fibers until they atrophy; in such cases the lens will tremble when the eye or the head is moved. Rupture of the fibers favors dislocation of the lens.

Sometimes the anterior capsule thickens and becomes opaque; this happens in the pupillary area and forms a white membranous cataract. Cholesterin crystals or calcium salts are sometimes deposited in the lens; they are seen as white points; this is called chalky cataract.

Morgagnian cataract forms when the cortex becomes so fluid that the nucleus settles into the most dependent part of the capsular sac (see Fig. 201). The semicircular border of the nucleus can sometimes be seen in the pupil, provided the surrounding liquid is sufficiently clear. Some vision may be had between this border and the margin of the pupil, in the same way that a dislocated cataractous lens leaves a part of the pupil clear. In young patients the nucleus may be absorbed, and then vision can be reinstated with an appropriate lens correction.

A hard cataract and a black cataract may be difficult to differentiate. The latter is due to an unusual degree of sclerosis or hardening of the whole lens. It is not actually cataractous, because the cortex has not undergone the changes that lead to cataract formation. Yet it has a similar effect on vision, and the indications for removal are the same.

Treatment

Indications for the treatment of cataract are to remove an obstruction to the vision, or to form a black pupil for cosmetic purposes. Surgical intervention affords the only certain means for meeting these indications.

Since senile cataract is more prevalent than all other classes combined, its management will be considered first. There will then be less difficulty in understanding the management of other varieties.

The patient is not usually seen by the surgeon until vision is greatly reduced. By this time the lens is so opaque that nothing can be seen behind it. Diseases of the vitreous, the retina and the choroid can only be suggested by the results of a subjective examination. No other hope for restoration of vision than that which may be obtained by operation can be given. The surgeon is not justified in promising more than to use his best judgment and skill in the undertaking.

Where the fellow eye is also cataractous, no advantage may be had from examining it. But where the media of that eye are clear, the condition of its various structures can be judged by their appearances. When these are favorable, a more hopeful operative prognosis can be given.

The first question concerns the function of the retina. The patient is seated facing a lighted window with the better eye occluded. He should be able to detect the shadows of moving fingers as they pass in front of the eye, or to count fingers as they are held before the eye; the farthest distance at which they can be counted is noted.

The room is then darkened. A lighted candle or the light from an electric ophthalmoscope or other small instrument is moved about before the eye. The patient should be able to point to the light in each of several locations. This is "projection." Two lights are placed together at a few meters distance and gradually separated. The patient is requested to state when he detects the presence of two lights. Where some of these tests are positive, an improvement will accrue from operation.

The second question concerns the operability of the eye and the operative risk. Since cataract results from faulty nutrition of the lens, and faulty nutrition depends upon some antecedent disease, no acquired cataract should be regarded as an uncomplicated condition. Cataract may complicate glaucoma or glaucoma may complicate cataract. In either case the tension of the eye will be raised, and preliminary measures must needs be used to reduce the tension before performing extraction. An old iritis or iridocyclitis with or without synechiæ may complicate the situation.

Opacities in the cornea or irregularities of its surface might defeat the objects of the operation. Central opacities require special consideration in the location of an iridectomy. Irregularities of surface contour would diminish the usefulness of the eye after removal of the cataract. Even then the patient might be able to go about alone, although denied the ability to read.

The extraction of cataract requires that the eyeball be opened, and infec-

tion becomes a possibility. Proper preliminary treatment must be instituted to free the patient of infections about the skin and the margins of the lids, conjunctivitis, trachoma, keratitis, corneal ulcers and infections of the tear passages. The presence of pathogenic organisms may be determined by placing a sterile eye pad over the eye for a night. The pad will collect secretions which may be examined in approved ways.

Epiphora suggests tear sac disease. Pressure should be made over the tear sac from the nose toward the eye; the expressed contents can be examined in the laboratory. Should no fluid be found by expression, it is advisable to dilate one canaliculus and inject several drops of half strength normal salt solution into the lacrimal sac. This should be expressed and submitted for laboratory examination. In any case of doubt, cataract extraction should be preceded by sealing the puncta with galvanocautery, ligating both canaliculi, or extirpating the tear sac. The Toti-Mosher operation does not protect the opened eyeball from infection.

Endogenous infections may exist. Oral sepsis, diseases of the respiratory and aural mucous membranes, and infections of the gastro-intestinal and genito-urinary tracts should be corrected.

Some surgeons regularly drill their patients to "look down," to "close the eye gently" and to "look up." This should be done only so far as to impress the patient that he must be alert and understand what is said to him. The patient must not speak during the operation except to answer questions.

After eliminating factors of danger the patient is sent to the hospital. A calm patient may be admitted the day before the operation. A nervous one should be sent in some days ahead of time.

The time of day for performing the operation is to be decided by the surgeon. Some operators prefer late afternoon because the coming night promises sleep and rest for the patient. Others prefer early morning hours after they themselves have had a night's rest and are refreshed.

Surgical Preparation of the Patient.—On the night preceding the operation the lashes of the upper lid should be trimmed. If the scissors are moistened with water or oil the cut lashes will cling to the blades and not fly about. The lashes on the nasal half of the lid may be left uncut if they are not very long; these can be used in emergencies for lifting the lid. Those on the temporal side should be cut short.

The eyelids, lashes and eyebrows should be carefully washed with bichlorid of mercury solution 1:5,000 or with ethereal soap and water. The latter should be rinsed off. The same surfaces should then be anointed with White's ointment, and, after instilling one drop of 2 per cent atropin sulphate solution, a sterile eye pad must be applied. Sodium bromid 1.3 gram (grains xx)

combined with chloral hydrate 0.65 gram (grains x) is to be given at bedtime. This dose should be repeated one hour before operation; no food is to be given that morning. Morphin is not given because it might cause nausea or vomiting.

The bowels should be cleansed by laxative and enema the day before the operation. They will need no attention for two or three days afterward. The patient must not strain at stool.

Supplies for the Operating Room.—Sterile water for making fresh solutions. Four 0.065 gram (grain i) powders of flake cocain for making solution. Novocain. Adrenalin chlorid solution 1: 1,000. Sterile normal salt solution. Bichlorid of mercury solution 1: 5,000. Atropin sulphate solution 2 per cent, fresh and sterile. Eserin solution 1: 500. Two strips of adhesive tape 1 centimeter (3/8 inch) wide and 15 centimeters (6 inches) long; and one strip 1 centimeter (3/8 inch) wide and 20 centimeters (8 inches) long. Four sterile eye pads. Sterile cotton, sponges and gauze. Alcohol for sterilizing instruments. Sterile water for rinsing instruments.

General Preparations.—Surgical principles must be observed, but the surgeon shall wear no gloves.

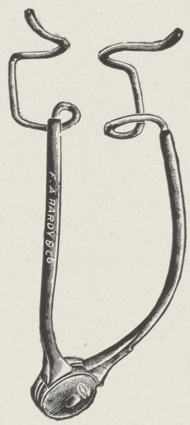


Fig. 203.—Smith's Speculum.

Fig. 204.

Fig. 205.

Fig. 206.

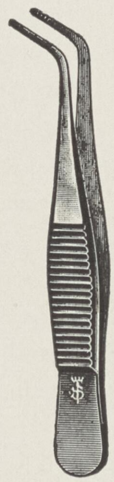


Fig. 207.—Fuchs' Capsule Forceps.

Instruments.—These are selected in two groups; one is made up of instruments that will be required, and the other of instruments to be used in emergencies. The required instruments are a spring speculum (Fig. 203), two

fenestrated lid retractors (Fig. 204), fixation forceps without catches (see Fig. 167), von Graefe cataract knife (Fig. 205), iris forceps (Fig. 206), iris scis-

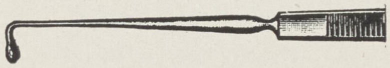


Fig. 208.

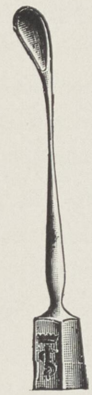


Fig. 209.

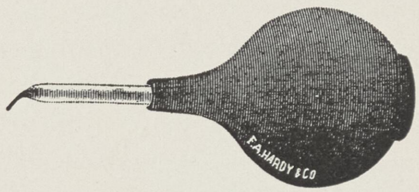


Fig. 210.

sors (see Fig. 169), lid hook (see Fig. 6), capsule forceps (Fig. 207), Smith's pressure hook (Fig. 208), spoon (Fig. 209), irrigator (Fig. 210), spatula (see Fig. 171), and iris repositor. Emergency instruments are extra knife and iris scissors, cystotome (Fig. 211), knife needle (Fig. 212), fixation forceps without catch and vectis or lens loop (Fig. 213).

Fig. 211. Fig. 212.

Fig. 213.—Lens Loop.

They are to be immersed in alcohol. Immediately preceding the operation they are transferred to sterile water, and dried. Neither points nor edges of cutting instruments shall be allowed to come in contact with anything unnecessarily. The instruments are arranged in two rows. Those known to be needed are to be laid out from left to right on the side of the instrument tray nearer the surgeon, and in the order in which they will be required. The emergency instruments are to be arranged in the farther row.

Anesthesia is effected almost wholly by cocain. A 0.065 gram (grain i) powder is dissolved in seventeen drops of sterile water and three drops of adrenalin chlorid solution 1:1,000 are added. One drop is instilled into the eye every two minutes until anesthesia is complete. It is of advantage to instill two or three drops into the fellow eye to abolish the winking reflex.

Novocain is employed to guard against one of the greatest dangers that can be encountered in cataract work, and that arises from squeezing of the lids over the opened eyeball. The orbicularis oculi should be paralyzed. Five drops

of adrenalin chlorid solution 1 : 1,000 are added to 2 cubic centimeters of 2 per cent freshly prepared solution of novocain. This is injected deeply into the extra-orbital tissues from the junction of the lateral and inferior margins of the orbit; part of the solution is directed toward the ear and part toward the ramus of the mandible. Some operators also inject a portion along the lower border of the lower lid. The infiltrations should be made fifteen to twenty minutes before opening the eyeball.

Cocain anesthesia is then obtained. A few drops of novocain solution are injected beneath the conjunctiva above the cornea. Immediately before operating, the conjunctival sac should be thoroughly irrigated with 1 : 5,000 solution of bichlorid of mercury. To assist in introducing the syringe into the fornices the lids are elevated with a lid hook, but that instrument must be sterilized again or laid aside during the operation. A 60 cubic centimeter (2 ounce) soft-tipped ear and ulcer syringe is useful in making the irrigation.

Some surgeons use trained assistants. Any one can assist if he appreciates the anatomy of the lids and orbit and understands the steps of the operation. It is essential that the assistant shall give *undivided attention* to his duties.

The Operation.—The operation proper is divided into five stages: The section, the iridectomy, the capsulotomy, the lens delivery and the toilet.

The Section.—The speculum or the retractors are used to hold the lids apart and away from the eyeball. No pressure shall be made on the globe.

The cornea is to be compared to the face of a clock on which the hour of twelve is located at the junction of the superior nasal and temporal quadrants of the limbus. In making the incision, a right-handed operator stands at the head of the patient when operating on the right eye, and at the left side when making the section on the left eye. If the surgeon is ambidextrous he may properly stand at the head of the patient when operating on either eye; in that case he will guide the knife with his left hand when making the section on the left eye.

The knife is taken in the fingers of the right hand. The thumb rests on that face of the handle which corresponds to the cutting edge of the blade. The tips of both the first and second fingers rest on that face of the handle which corresponds to the back of the blade. The section is made almost entirely by movements of the fingers. The knife is held lightly and delicately, with the cutting edge always directed toward twelve o'clock.

The patient is directed to look upward. The fixation forceps are closed and applied to the bulbar conjunctiva at five o'clock for the right eye, and at seven for the left, as near the cornea as it is possible to pick up conjunctiva. The closed jaws are pressed against the eyeball and opened; this strips the blood out of the vessels. A fold of conjunctiva is then picked up evenly and

the forceps are rotated a quarter turn to twist the fold; the twist assures a firm hold to prevent the eyeball from turning. The forceps must *not press* on the globe. If the conjunctiva tends to roll over the nasal margin of the cornea the twist should be reversed.

The extent of the section is determined by the estimated size of the lens nucleus. The point of the knife is applied to the margin of clear cornea, or to the sclerocorneal junction as desired, at nine-fifteen o'clock for the right eye and at two-forty-five for the left, or about 1 millimeter above the horizontal meridian of the cornea (Fig. 214) in the average case. The handle of the knife is elevated slightly so that the point engages at an angle to the surface of the cornea.

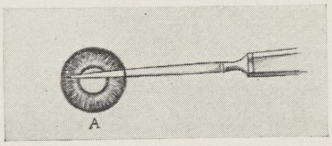


Fig. 214.—Puncture.

The point is directed toward the jaws of the fixation forceps and gently thrust through the thickness of the cornea. The blade must pass through the cornea and not merely split the lamellæ. When the point is within corneal substance it is dull gray in appearance, while in the anterior chamber it is bright.

The handle is then lowered and the blade is gently pushed by the fingers two-thirds of the distance toward the fixation forceps and parallel with the plane of the iris. The latter must not be injured. The handle is now pushed downward (Fig. 215) until the blade is parallel with the horizontal meridian of the cornea. The counterpuncture is made at two-forty-five o'clock for the right and at nine-fifteen for the left eye, opposite the puncture. The point of the knife should be directed about 1 millimeter nearer the surface than the point intended for the counterpuncture, or the latter will be made too deeply. When the point of the knife appears outside the eye the section can be made.

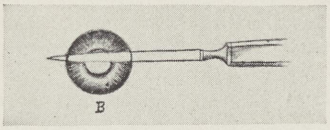


Fig. 215.—Counterpuncture.

The cornea should be divided parallel with the plane of the iris. The point

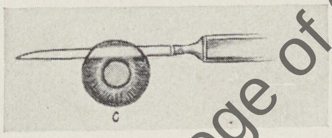


Fig. 216.—Section.

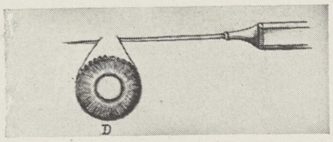


Fig. 217.—Conjunctival Flap.

of the knife is thrust nasally (Fig. 216) and upward for the length of the blade and then drawn temporally and upward an equal distance. The section is completed by a to-and-fro or sawing motion; the blade must always cut in its own plane. Should the iris catch on the knife, the section must be completed.

When the cornea has been divided the conjunctiva which lies in front of the edge of the knife is divided by another saw movement so as to form a small flap (Fig. 217).

The Iridectomy.—The operation is called a *simple extraction* when no iridectomy is made, and a *combined extraction* when an iridectomy is performed. The advantage of a simple extraction is that it leaves a round pupil. The aqueous escapes and the cornea collapses when the section is made; the pupil usually contracts regardless of how thoroughly it has been dilated. The delivery of a large nucleus through a small pupil might injure the iris, and there is danger of a secondary iris prolapse into the wound without iridectomy.

The surgeon stands at the head of the patient with the iris forceps in one hand and the iris scissors in the other. The assistant elevates the upper lid

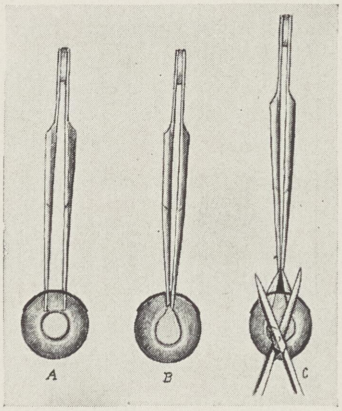


Fig. 218.—Narrow Iridectomy.
(After Meller.)

with a retractor or lid hook and draws the lower lid down with the thumb of the other hand. Or the surgeon may elect to use the speculum. The patient is directed to look down. The closed forceps are gently inserted between the lips of the wound to the pupillary border of the iris (see Fig. 218, *A*). The blades are slightly separated, and by pushing backward a little the iris will usually come up between the jaws of the forceps (see Fig. 218, *B*). The iris is grasped and gently drawn a little way out of the wound.

A narrow coloboma is desirable. The opened scissors are held parallel with the vertical meridian of the cornea (see Fig. 218, *C*), and the iridectomy is made with a single snip of the scissors close to the sclera. Where glaucoma exists a cut is made on each side of the forceps, and the section of iris is torn from its root. A wide coloboma is made by holding the scissors parallel with the palpebral aperture and cutting the iris horizontally in the direction of the corneal incision (see Fig. 173).

Some operators choose to make a peripheral iridectomy or a buttonhole in the periphery of the iris. In such cases the iris is grasped at the periphery and not at the pupillary margin. Sometimes the iris is degenerated or sclerosed so that no hold may be obtained with the forceps. The iris hook is used to draw the iris out of the wound. Having made the iridectomy, the iris repositor is swept between the lips of the wound from each angle of the incision to the center, to clear it of iris tissue and to replace the cut edge of the iris in position (Fig. 219).

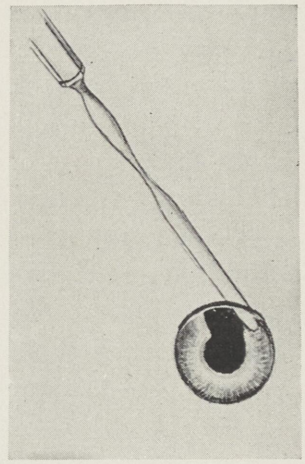


Fig. 219.—Repositing Iris. (After Meller.)

The capsulotomy is made with the capsule forceps, the cystotome or the knife needle. The surgeon elevates the upper lid with a lid hook, the assistant pulls the lower lid down with his thumb, and the patient is directed to look down. The object of a capsulotomy is to provide an opening in the capsule through which the lens can be delivered.

The forceps are introduced closed between the lips of the wound until the teeth are well within the pupillary area. The jaws are allowed to separate, but not enough to catch the iris (see Fig. 220, *A*). The teeth are pressed against the lens only enough to bite into the capsule. Some surgeons gently shake the forceps to loosen the lens. The forceps are closed and withdrawn with a piece of the capsule (see Fig. 220, *B*). Should the forceps fail, other methods may be used.

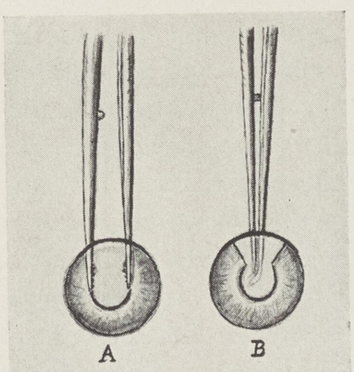


Fig. 220.—Capsulotomy. (After Meller.)

The cystotome is introduced with the cutting point parallel with the plane of the iris, and then turned back. The capsule may be opened by an inverted "V" incision or by a crucial incision. The instrument must not be sunk into the substance of the lens, or the latter may be dislocated. The knife needle is used in the manner of the cystotome in cases where the capsule is so thickened or tough that it cannot be torn without undue force if the cystotome were used.

The Delivery of the Lens.—The assistant separates the lids and raises them off the globe with the retractors. The surgeon holds a lens spoon in one hand and an expression hook in the other. The spoon is held with its convexity toward the eyebrow and one edge lightly touching the scleral lip of the inci-

sion. In this position it is ready to depress the scleral lip when the upper edge of the lens presents, or to move behind the lens if vitreous presents.

The tip of the hook is applied to the cornea at six o'clock with the direction of pressure toward the center of the globe. By pressing intermittently, the upper border of the lens is induced to engage in the capsulotomy wound. Then by directing the hook pressure somewhat upward and indenting the cornea below, the lens is followed until it passes through the corneal incision. It is caught on the spoon or in the elbow of the hook and carried clear of the operative field. The retractors are removed and the lids are closed.

The delivery should be conducted without haste. Sometimes the lens holds tenaciously, and at other times it slips out readily. The section should be large enough to permit the passage of the lens without binding. On account of the curvature the width of the section on the posterior surface of the cornea is some 3 millimeters less than on the anterior (Fig. 221).

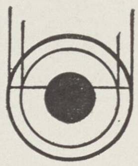


Fig. 221.

The Toilet.—The upper lid is elevated with a lid hook. The patient does not look down. Cortical débris and capsule fragments are washed out by irrigating the anterior chamber with a stream of normal or half normal salt solution. A gravity irrigator or a soft-rubber syringe may be used. In either case a fine cannula is fixed solidly to the apparatus. The tip of the cannula is inserted merely between the lips of the wound at each angle alternately. Air bubbles do no harm.

Where irrigation fails, slender forceps may be inserted to remove particles. Delicate manipulations are required to avoid such complications as a loss of vitreous. It is better to leave material in the anterior chamber than to excite an inflammatory reaction or any other complication by overzealousness.

The iris repositor is employed to free the wound of iris tissue and fragments of lens matter. The angles must be swept, for it is here that particles of lens or iris may be firmly impacted. The instrument is carried toward the center of the section from each angle (see Fig. 219). The iris should lie back in the anterior chamber, and when the light is strong enough the position of the coloboma may be inspected. The upper lid is lowered on to the globe.

One drop of 2 per cent solution of atropin is instilled if indicated. After simple extraction one drop of 1 : 500 solution of eserin is sometimes instilled to prevent secondary prolapse of the iris. It is seldom indicated with iridectomy.

Dressings.—White's ointment is applied along the external surface of the lines of lashes to prevent adhesion of the dressings. Pads of cotton or gauze slightly smaller than the orbital opening are saturated with 1 : 5,000 solution of bichlorid of mercury. Several of these are placed smoothly over

the treated eye, and one over the untreated eye. A sterile eye pad is moistened with the same solution and placed over the treated eye. Dry sterile pads are placed over both eyes. The dressings are secured by strips of adhesive (see Fig. 8). A 20 centimeter (8 inch) strip is placed across both eyes and the nose toward the ears.

In some localities it is customary to swathe the head in gauze bandages. A variety of protecting masks and shields are available for those who wish to use them.

The patient is lifted from the operating table to the cart, unless the cart was used as an operating table, and at his room he is lifted from the cart to his bed where he is to lie quietly on his back for five to six hours. Instructions are given that he may lie on his back or on the side of the untreated eye. If delirium develops, the dressings are to be removed from the untreated eye and bromids are to be given. Complaints of pain and the appearance of discharge must be reported immediately to the surgeon.

After Care.—The patient is to remain in bed forty-eight hours without disturbing the dressings. Liquid diet is given during this period. At the end of forty-eight hours, the third day, the dressings are removed, the edges of the lids are gently cleansed with 1 : 5,000 bichlorid of mercury solution, the lower lid is drawn down and one drop of 2 per cent solution of atropin is instilled. Eye pads moistened with bichlorid solution are applied and secured as before.

On the fourth day the patient may sit up an hour or two both morning and afternoon, and have soft diet. On the fifth day atropin is instilled, and the dressings are renewed on the treated eye only. General diet is given. Daily thereafter, dressings are made after instilling atropin solution. The patient may be permitted to walk about a little, but with an attendant.

The patient is dismissed from the hospital after ten days. He is instructed to remove the pad each morning and wash the lids with hot water. If the eye does not ache or show considerable redness, atropin is to be used once daily, otherwise twice. Dark glasses may be worn after one week and the patient may go out of doors. When the eye is clear the atropin and dressings may be omitted. Glasses should be fitted in one month.

Complications of cataract extraction are numerous, but happen in few cases. When the tension is raised the vitreous may prolapse or intra-ocular hemorrhage of the expulsive type may occur. Low tension usually signifies thin or fluid vitreous; in such cases collapse of the eyeball is possible. Some iridectomies are accompanied by a hemorrhage that fills the anterior chamber. When the blood is allowed to clot it may be removed by gentle irrigation. Conjunctival hemorrhage occurs when the section is made too far back in the sclera.

Vitreous Prolapse.—This accident is imminent whenever the pupil becomes intensely black. It usually dilates also. A preliminary capsulotomy may prevent loss of vitreous. It is usually done on the day preceding the major operation. The local preparation of the patient is the same as for cataract extraction. Anesthesia is by cocain. The instruments needed are speculum, fixation forceps and knife needle.

The conjunctiva is grasped with fixation forceps at six o'clock. The needle should pierce the cornea at the point selected for the puncture at the operation next day. The knife enters the anterior chamber flat, with the cutting edge directed toward twelve o'clock. When the blade lies over the pupil, the edge is turned toward the lens. The capsule can be opened by one of the incisions already mentioned.

In cases of raised intra-ocular tension or squeezing of the lids, the vitreous may prolapse or the lens may be expelled entire as soon as the section is completed. A preliminary capsulotomy favors spontaneous delivery of the lens. The escape of aqueous and the lens may lower the tension sufficiently to prevent prolapse of the vitreous. The lid hook should supplant all other instruments for lid control.

When it is seen that vitreous loss is imminent, the lids should be closed promptly. The patient is ordered to avoid looking down. With the lid hook the lid is drawn forward off the eyeball, and this diminishes the prospects of vitreous prolapse.

In cases of complicated cataract, a preliminary iridectomy may be made about six weeks prior to the extraction. In cases of hypertension the iridectomy should be made wide and extend to the root of the iris. A trephining may be performed below at six o'clock, so as to be entirely removed from the later operative field.

Several sutures have been devised, not only for the purpose of safeguarding against loss of vitreous, but also to bring the edges of the incision in better coaptation for accurate healing. Kalt's suture and Berens' untied suture illustrate the idea. For the former, a fine silk suture is passed vertically through half the thickness of the cornea immediately within the limbus at twelve o'clock, and then horizontally through the conjunctiva and episclera some 5 or 6 millimeters above the cornea.

The loop of silk is laid over toward the nose so that it will be out of the way of the section. The lower free end of the thread is laid over the cheek, and the upper over the brow. Having closed the wound by traction on the upper thread, the suture is tied at the completion of the operation. It is removed on the third day. This also serves to guard against delayed loss of vitreous.

Berens' suture is employed in the following manner: The section is made and includes a broad bridge of conjunctiva. The suture is introduced as shown in Figure 222. The conjunctival bridge is divided with scissors at the point indicated by the dotted line. The iridectomy, capsulotomy, lens delivery and toilet are completed as usual. Then the suture is drawn tight, and each end is laid along the canthus nearest it; the lids are closed and the dressings are applied. The suture is removed in three to five days.

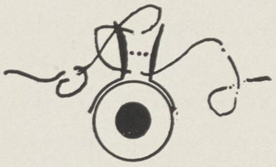


Fig. 222.

A Van Lint sliding flap of conjunctiva is sometimes made to serve the same purposes as the sutures. Two sutures, one at either side of the cornea, bring the apron flap down over the corneal incision at the completion of the operation (see Fig. 100). These may be removed in five or six days.

An external canthotomy prevents squeezing of the lids with a consequent loss of vitreous; it is preferable and just as effective to paralyze the orbicularis with novocain, unless the palpebral aperture is small.

Intra-ocular hemorrhage of the expulsive type is rare. It arises from suddenly relaxing the pressure on the weakened vessels of the choroid. The lens emerges spontaneously, and leaves an intensely black pupil. The vitreous follows the lens, and is followed by blood. Lastly, the retina may appear as a crumpled membrane between the lips of the wound. The eyeball is lost.

Dislocation of the Lens.—The lens loop or the spoon is employed to recover and deliver the lens. The shank of the blade may be bent in bayonet fashion and then slipped behind the lens. The latter is pressed forward against the cornea and gently lifted out without pressure against the posterior lip of the incision.

Collapse of the Cornea, or inversion of the convexity, sometimes accompanies the escape of the aqueous when the section is completed. The anterior chamber soon fills with aqueous, or it may be filled with half strength normal salt solution before applying the dressings.

After Treatment and Late Complications

Morphin and general anesthesia must be avoided whenever possible, since they may cause nausea and vomiting. All violent movements or straining must be avoided. No patient who has a cough should be subjected to a cataract extraction.

Many cataract patients are old and decrepit; they cannot be kept continuously on their backs nor have both eyes bandaged over a considerable period of time without possible delirium, or serious mental manifestations. It is

frequently advisable to prop them up in bed, and to remove the bandage or pad from the untreated eye in forty-eight or even in twenty-four hours. They may be placed in wheeled chairs on the third day without especial solicitude. An attendant should be constantly in charge.

The patient should have comfortable rest during the night following the operation. Some sedative such as bromids or veronal may be given.

Some individuals have a habit of unconsciously rubbing their eyes. It is permissible to guard the cataract patient by tying the wrists to the cot in such a manner that he cannot reach his face, while he has liberty of motion in other directions.

Panophthalmitis is the most serious complication that can follow the operation. The signs of infection appear on about the third day, when mucopurulent discharge is found on the dressings, or is seen coming from between the lids. Pain on the preceding night is suggestive unless accounted for otherwise. Where the infection is deep within the globe, little can be done. When it is superficial, it may be possible to save the eyeball and some quantitative vision.

The conjunctival sac should be cleansed, after local anesthesia, by irrigating it with 1 : 5,000 bichlorid of mercury solution. Where the point of infection is found in the incision, that point may be cauterized with phenol or the galvanocautery. The dressings should be kept off to permit drainage, and the sac should be irrigated frequently with antiseptic solutions. When the process gets beyond control, it must be treated as panophthalmitis from any other cause.

Iritis.—This occurs between the second and tenth day from endogenous or exogenous infections. Lens material left in the anterior chamber is sometimes exciting. Membranes form and block the pupil, and sometimes draw the pupil upward.

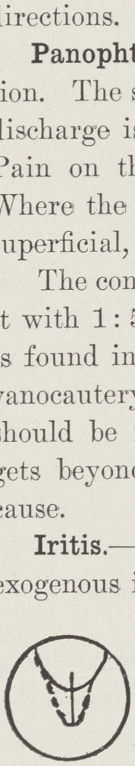


Fig. 223.

Vitreous prolapse and intra-ocular hemorrhage may be delayed in their appearance. The pupil is drawn up and the iris is crescentic in form (Fig. 223). These complications contribute to a prolonged convalescence.

Delayed Healing.—The retention of iris tissue, lens matter or vitreous between the lips of the wound tend to prevent healing. These are called inclusions or impactions. In few instances will the delay be caused by inability to heal. The main sign of delay will be found in the absence of a re-forming anterior chamber. A small leak may be located. Any protruding substance should be excised. The wound edges are freshened with a knife point, or painted with a 1 per cent solution of silver nitrate. A broad conjunctival flap may be brought down to cover the wound. Atropin is indicated. The dressings are left off, and moderate lid movements are permitted.

Prolapse of the Iris is an occasional complication, particularly following a simple extraction. Rupture of the healing wound allows an outward flow of aqueous, which carries the iris with it. Retained lens material may swell and crowd the iris into the incision. Iris tissue may have been left in the wound at the conclusion of the operation; it would afford a pathway for infection, or it might lead to secondary glaucoma.

When the prolapse is not reduced by atropin it requires surgical treatment. The lid hook is used to hold the lid away from the globe and to guard against the escape of vitreous. The prolapse may be removed by cautery, or the protruding portion may be grasped with fine forceps, drawn out of the wound a little, and cut off as closely to the original incision as possible. The new wound should be cleared of iris tissue with a spatula, and covered with a conjunctival flap. Atropin is instilled and dressings are applied daily until healing is completed.

When the conjunctiva is unhealthy and the prolapse is causing no particular irritation, haste in operating is not essential. Yet the earlier it is done the more easily it can be performed.

Prolapse of the Vitreous may occur during the convalescent period. The wound fails to close and the symptoms are those of ocular irritation. It affords a pathway for infection, and glaucoma may follow. Where an inflammatory reaction occurs, a band from the inclusion may extend into the vitreous body and produce a form of secondary cataract to obstruct vision. This band may have the effect of drawing the pupil upward so that the remaining iris has the form of a crescent.

Treatment of this condition is designed to prevent infection, to reduce the vitreous hernia, and to quiet the iris. The conjunctiva is detached from the temporal side of the cornea; a parallel incision is made 6 millimeters from the first. The strip is then undermined. The prolapse is excised, and the flap is drawn over the cornea and secured in place; the denuded sclera is covered by drawing the edges of the intact conjunctiva together and over it by one or two sutures. Atropin is instilled. Irritation of the fellow eye suggests sympathetic ophthalmia.

Impaction of Capsule Fragments.—The symptoms are those of ocular irritation. Iridocyclitis or glaucoma may result. A localized appearance of keratitis develops in the area of the impaction. The capsule is epithelial in nature and it invites a growth of corneal or conjunctival epithelium into the wound. Should this epithelium cover the spaces of Fontana, glaucoma will surely result. A cyst of the anterior chamber may form.

The dressings are removed in order that the lids may sweep the surface of the wound and possibly dislodge the fragment. Sometimes the fragment

can be removed with forceps. At other times it may be best to open the wound, curette the edges, and cover it with a conjunctival apron.

Masses of lens cortex remaining in the anterior chamber may swell and produce glaucoma; this is often of a progressive type and requires treatment.

Detachment of the Choroid may be suspected when the globe is very soft, but its presence cannot be certainly determined without an ophthalmoscopic examination. A completely formed but very shallow anterior chamber is usually present. The complication is corrected spontaneously as a rule.

Entropion of the spastic variety demands removal of the dressings. The lid may be held in eversion by a strip of adhesive tape, or a temporary suture may be used.

Other Operations for Removing Cataract

Intracapsular Extraction of cataract, or *expression* of the whole lens, is practiced by some operators in order to avoid the complications which may arise from retained fragments of lens and capsule.

The leading points of two procedures will be given: The expression method popularized under the name of the Smith-Indian operation, and the suction method, also known as phaco-eresis or Barraquer's operation. In both methods it is unnecessary to use atropin, since an iridectomy but no capsulotomy is performed. The corneal section is made well back in the limbus to afford room for the delivery of the lens in its capsule. The knife crosses the anterior chamber deeply. A conjunctival flap of moderate size should be included in the section.

The Smith-Indian Operation.—The iris forceps are introduced into the wound and opened slightly. If the shank of the forceps is pressed against the scleral lip of the incision, a fold of iris will usually rise between the jaws of the forceps. The iridectomy is completed.

The assistant raises the upper lid forward off the eyeball with a lid hook held in one hand, and depresses the lower lid with the thumb of the other hand against the lower orbital rim. The surgeon holds an expression hook in his right hand, and in his left a lens spoon. The spoon is held concavity forward, all but touching the edge of the scleral lip of the incision. The hook is then applied to the lower area of the cornea.

The lens is delivered in one of two ways. It may be delivered upright, or it may be tumbled; for the latter method the lower border of the lens is made to travel through an arc forward against the posterior surface of the cornea until it presents in the wound.

Accidental rupture of the capsule sometimes occurs. It is desirable to deliver the capsule and lens together, and for that purpose the mass may be

grasped with toothed forceps. If this plan is unsuccessful, capsular fragments may be removed as in combined extraction.

The toilet of the wound is important. All iris tissue must be removed from between the lips of the incision. The use of atropin is not advised after expression. The usual dressings are applied, and the patient is guarded against any exertion.

Barraquer's Operation requires a special apparatus. The section and iridectomy are made as before. The closed end of a metallic tube is fashioned into a hollow spoon. An aperture is made in the concave plate of the spoon, and the tube is connected with a special vacuum pump. The concavity of the spoon is applied to the anterior surface of the lens. A vibratory movement loosens the zonular attachments. The lens is well fixed, by suction, to the spoon, and is delivered by being drawn up and out against the posterior surface of the cornea. In this operation the patient does not look down during the delivery.

Linear Extraction.—This operation is applicable to soft cataracts, in which the nucleus is neither large nor dense, and particularly in patients below the age of thirty years. It is also the operation of choice in cases of traumatic cataract, or in intumescent cataract of rapid development where acute glaucoma is a factor of importance. Owing to the youth of the patient, general anesthesia may be advisable. The usual preparations are made and the pupil is dilated with atropin. The instruments are the same as for ordinary extraction, except that the section may be made with a keratome.

The globe is held with fixation forceps. The point of the keratome is applied to a point about 2 millimeters down from twelve o'clock. The blade is directed toward the upper margin of the pupil to avoid splitting the corneal lamellæ. An alternative method is to start the incision in the limbus at twelve o'clock, and to push the instrument downward in a plane parallel to that of the iris. The keratome should be made to cut with one edge at a time.

Where it is designed to perform a capsulotomy, the point of the instrument can be made to engage and rupture the capsule; otherwise the point is elevated toward the posterior surface of the cornea enough to avoid both lens and iris. In some cases of traumatic cataract the capsule is already broken. By slightly rocking the blade, much of the lens substance will escape with the aqueous. When necessary to rupture the capsule, it should be done with a cystotome.

The lens matter is delivered by making light pressure on the scleral lip of the wound, and gently massaging the cornea with a lens hook or spoon. The anterior chamber may be irrigated with warm normal salt solution, or half strength, for removing fragments of lens. Atropin is applied and both eyes are

dressed. The dressings may be omitted from the untreated eye after twenty-four hours, and from the treated eye as soon as the section has healed, usually in five or six days. Dark glasses should be worn.

Complications.—Iritis is an occasional complication, and atropin should be continued for some weeks. Vitreous prolapse is indicated by a pupil which suddenly becomes black. Since soft cataracts are found in defective and injured eyes, it is wise to be conservative about removing every vestige of lens substance; much of this will be absorbed by the action of the aqueous.

Discission.—This operation also is used for the removal of soft cataracts, especially in the very young or those up to sixteen years of age. It has to be repeated an indefinite number of times to effect complete absorption of the lens. The preparation is the same as for simple extraction. The instruments are speculum, fixation forceps and knife needle.

The fixation forceps grasps the conjunctiva close to the limbus, opposite the point elected for introducing the needle. The needle is held with the flat side of the blade parallel with the plane of the iris. The point is passed beneath the conjunctiva for 2 or 3 millimeters before it is made to enter the anterior chamber. When the blade lies over the center of the pupil, the cutting edge is turned toward the lens. Vertical and horizontal incisions of not more than 3 millimeters in each direction are made to cross over the center of the capsule (Fig. 224) without disturbing the cortex. The blade is turned on the flat and withdrawn without force. Very little or no aqueous should be lost.

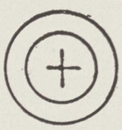


Fig. 224.

Atropin and White's ointment are applied, and the eye is covered with a patch. Unless atropin is continued until the reaction subsides, synechiæ may form. When all signs of reaction have disappeared, a second needling is done. The first only incises the capsule. Subsequent ones must deal with the cortex and nucleus.

Intumescent cataract may arise from a too free opening of the capsule at the first operation. This leads to rapid swelling of lens substance, the iris is crowded against the filtration angle, and an acute glaucoma results. It may become necessary to perform a linear extraction.

After-Cataract.—This often follows the removal of a cataractous lens, and results from a variety of causes. Infection produces an exudate which may undergo organization, retained lens fragments may not be absorbed, capsular epithelial cells may proliferate, and the posterior capsule may thicken or wrinkle. Each of these will occupy the pupillary area and obstruct vision. These are sometimes classed as membranous cataracts.

In the treatment of this condition the opaque mass must be divided. The knife must penetrate into the vitreous, and the latter tends to follow the blade

as it is withdrawn and adhere to the corneal wound. Its presence prevents healing, and forms a pathway for infection.

Needling (or Discission).—The incision is usually made at or near the limbus. The knife needle is passed into the anterior chamber as for discission, and is then turned into the cutting position. An inspection of the opacity may reveal a striation in one definite direction. It is advisable to make the incision at a right angle to the striations. In such cases a single division of the fibers is usually sufficient; they retract and form an oval pupil (Fig. 225). When an adequate aperture is not formed by a single incision, a second one must be made.

Fig. 225.

Where two incisions are required, an inverted V or an upright T (Figs. 226 and 227) usually allows the flaps to turn or roll downward in a manner that leaves a useful pupil. The needle is turned on the flat and withdrawn quickly. Atropin is used cautiously. Needling is not performed until the eye is free from signs of congestion, and is quiet so far as iritis and cyclitis are concerned.

Fig. 226.

Fig. 227.

Iridotomy.—This may be the operation of choice where the pupil has been drawn high up in the anterior chamber. A narrow-bladed knife is thrust through the limbus into the anterior chamber, until the point is somewhat to the medial side of the vertical meridian of the iris and well down. The point is then pushed through the iris and any membrane behind it, and a cut is made upward into the pupil. The membranes usually retract and leave a slit pupil (see Fig. 223).

Nonsurgical treatment of cataract: Various methods and agents have been devised and advised for this purpose. Lens extract has given promise in the hands of some physicians, while it has been tried and abandoned by others. Dionin, potassium iodid solutions, and subconjunctival injections of normal salt solution have been used.

Amber glasses are an asset to vision in early stages of cataract, since a quality of the glass is to sharpen the definition of images.

Dislocation of the lens is infrequent. There are two varieties: Luxation when no part of the lens occupies the pupillary area, and subluxation when it is partly or wholly within that area. Dislocation requires rupture of the zonular fibers, and this allows the lens to thicken anteroposteriorly, so that myopia results. When no part of the lens is in the pupil the visual effect is identical with that following extraction of cataract, and hyperopia results. Accommodation no longer exists.

The lens may be dislocated into the anterior chamber. The appearance is that of a drop of oil outlined by a yellowish circle. The anterior chamber

is deeper below than above. The lens should be removed. In some cases the lens lies in the pupil.

A partial dislocation may be detected with a plus 7 lens in the sight hole of the ophthalmoscope; one border of the lens is seen as a curved line inside the pupillary area (Fig. 228). On the concave side of the line the fundus is seen with one lens, while on the convex side of the line the lens used in the instrument must be about 10 diopters stronger. On account of this division in the pupil the patient may have diplopia. Astigmatism will be present also if the lens is tilted so that one edge is nearer the cornea than the other.

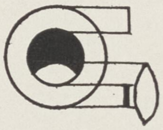


Fig. 228.

The lens may be dislocated into the vitreous, and the vision is affected as it is after extraction of cataract. It is usually best to leave such a lens alone.

In all known cases of lens dislocation it is advisable to watch for the occurrence of glaucoma.

CHAPTER XVI

THE EXTRA-OCULAR MUSCLES

ESSENTIALS OF THE ANATOMY

The eyeball is suspended in the orbit and held in position by the orbital fat, the extra-ocular muscles, the check ligaments and the fascial structure.

The position of the eyeballs and the direction of the line of sight of each eye, with relation to the head and surrounding space, is under the control of the six extra-ocular muscles. The four recti muscles arise from a tubular tendon at the apex of the orbit and are inserted into the sclera near the cornea. The superior oblique arises from near the tubular tendon, passes through its pulley near the superior medial angle of the orbit, and is reflected backward and outward over the globe. The inferior oblique arises from the inferior medial margin of the orbit and passes backward and outward under the globe.

The tendons of the recti are inserted at different distances from the cornea. According to Fuchs the distances average as follows: Medial rectus 5.5 millimeters, inferior rectus 6.5 millimeters, lateral rectus 6.9 millimeters, and the superior rectus 7.7 millimeters from the cornea.

The muscles must be studied collectively for the two eyes before precise binocular movements can be appreciated. All of the twelve muscles participate in every movement of the eyes. It is also necessary to understand axes and planes of rotation. The movements of the eyeball are executed about its center of rotation in the manner of a ball and socket joint. This center is located near the intersection of the anteroposterior, transverse and vertical axes.

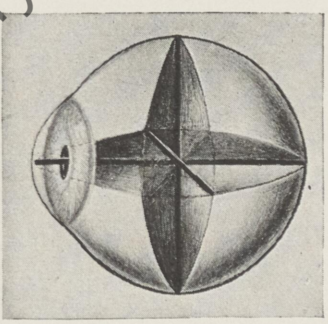


Fig. 229.—Principal Axes of Globe.

There are three *planes* of rotation (Fig. 229). The sagittal plane bisects the globe into lateral hemispheres; the frontal or equatorial plane bisects it anteroposteriorly; and the horizontal plane bisects it into upper and lower halves. The continuation of the sagittal plane forward defines the vertical meridian of the cornea. All rotations are calculated as having started from the primary position (Fig. 230). This position is attained when the head is held erect and the gaze is directed straight to the front on a horizontal plane

at some object situated at infinity. The visual lines of the two eyes are then parallel.

Listing's law substantially states that when an eye is turned from the primary position to a secondary position *it is rotated about an axis that is at*

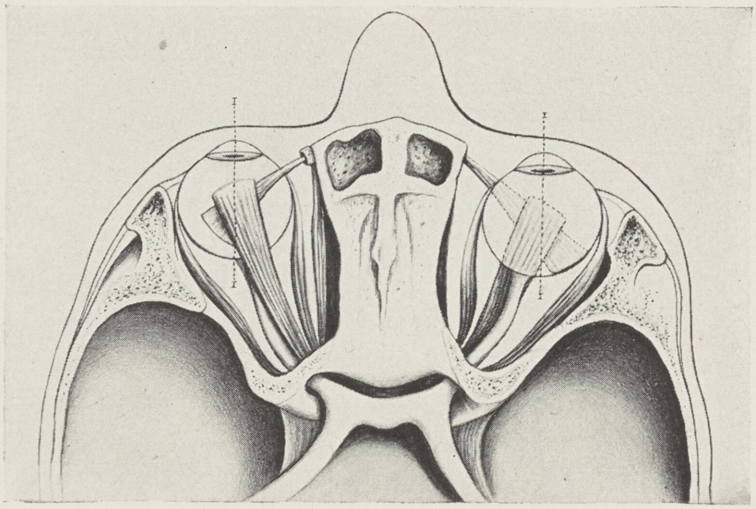


Fig. 230.—Primary Position. (After Landolt.)

once perpendicular to both the first and second positions of the visual line or anteroposterior axis. Listing's plane is the equatorial plane. Listing's axes are all contained in this plane; they are vertical, horizontal and oblique. For a given direct rotation the axis remains constant. Rotation refers to placing the visual axis in a desired direction, and this is indicated by the situation of the cornea with reference to the primary position; it is rotated to the right or left, up or down, or diagonally.

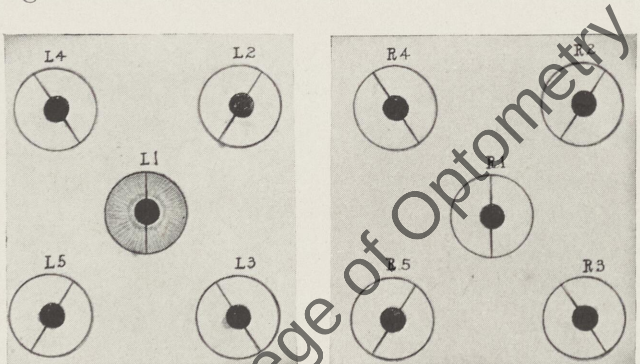


Fig. 231.—Rotations of the Vertical Corneal Meridians.
R, right eye; *L*, left eye. Numerals paired.

Duction is a turning of one eye in any direction. A rotation of the cornea toward the temple is abduction, a rotation toward the nose is adduction, a movement upward is sursumduction or elevation, and a movement downward is deorsumduction or depression of the cornea. An inclin-

ation of the vertical meridian of the cornea is called torsion; when the upper end is inclined in, it is intorsion, and when inclined out it is extorsion. Torsion is also called "wheel motion." Intorsion occurs when looking "up and in" or "down and out"; extorsion occurs when looking "up and out" or "down and in." While the meridian of one eye is intorted that of the other is extorted (Fig. 231).

A muscle can exert its maximum power when its "axis of traction" lies in or near a plane that passes through the visual axis. Its field of action is in the direction toward which it rotates the cornea or the visual axis when in its

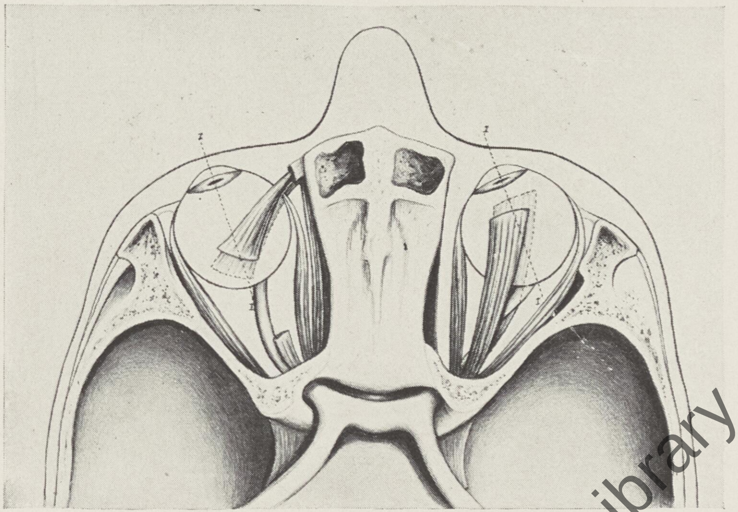


FIG. 232.—MUSCLES ACTING IN LOOKING TO LEFT.

most favorable position. Its associate action is synergistic to the predominant actions of other muscles.

The medial rectus rotates the cornea nasally. In this position the obliques can exert their maximum power in rotating the cornea "down and in" or "up and in" respectively. This muscle is assisted by the superior and inferior recti in maintaining nasal rotation (see Fig. 232, right eye).

The lateral rectus rotates the cornea temporally. In this position the superior and inferior recti can exert their maximum power in rotating the cornea "up and out" or "down and out" respectively. The lateral rectus is assisted by the obliques in maintaining temporal rotation (see Fig. 232, left eye).

The superior rectus forms an angle of 23 degrees with the sagittal plane of the globe. When the cornea has been moved temporally 23 degrees this

muscle is an elevator; it rotates the cornea upward. The levator palpebrae superioris lies between this muscle and the periorbita above. Their fascial sheaths are united posteriorly, which partially accounts for the elevation of the upper lid when the cornea is moved upward. The reflected tendon of the superior oblique lies between the superior rectus and the eyeball (Fig. 233).

The inferior rectus forms an angle of about 20 degrees with the sagittal plane of the globe. When the cornea is moved outward 20 degrees, this muscle

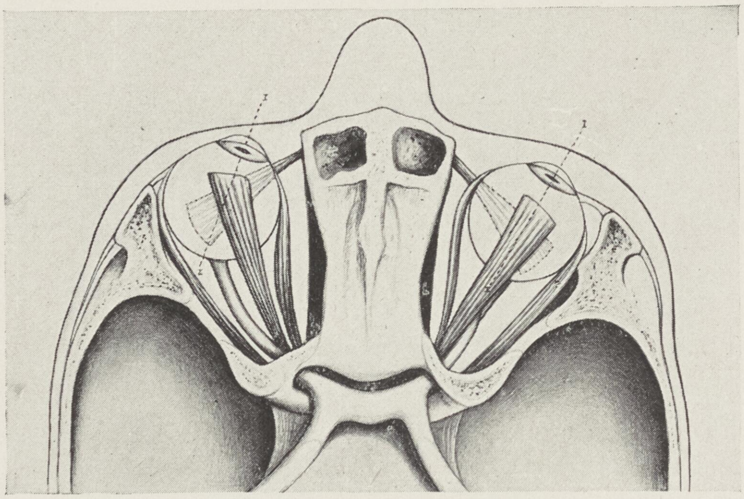


Fig. 233.—Looking to Right.
Ready to look up and to right.

is a depressor; it rotates the cornea downward. The inferior oblique passes beneath the inferior rectus; the fascial sheaths of these two muscles unite to form the suspensory ligament of Lockwood, which helps to support the eyeball in its position (see Fig. 234, left eye).

The superior oblique passes forward to the superior medial margin of the orbit where it becomes tendinous and passes through the trochlea or pulley. It is then reflected backward and outward over the globe. Its axis forms an angle of about 39 degrees with the sagittal plane of the globe. The tendon passes between the superior rectus and the eyeball, and is inserted into the sclera on the lateral side behind the equator. When the cornea is moved nasally this muscle is a depressor; it rotates the cornea downward. This action is shown in Figures 234 (right eye) and 245 (both eyes).

The inferior oblique arises from near the upper opening of the nasolacrimal canal. It passes backward and outward beneath the inferior rectus to be inserted into the sclera behind the equator on the lateral side of the globe. Its axis forms an acute angle with the sagittal plane of the globe. When the

cornea is moved nasally this muscle is an elevator; it rotates the cornea upward (see Fig. 233, left eye).

Each muscle is covered by a thin fibrous sheath. The check ligaments are formed of fibers which extend from these sheaths to the anterior margin of the orbital walls. They limit the rotations of the globe. Some fibers are inserted into subconjunctival tissues so that the fornices are retracted when the eyeball is rotated. It must be emphasized that ocular rotations are limited in range because they are concerned with fine adjustments. The neck muscles take care of coarse adjustments by placing the head in favorable positions. Torticollis imposes an inconvenience in changing the direction of the gaze.

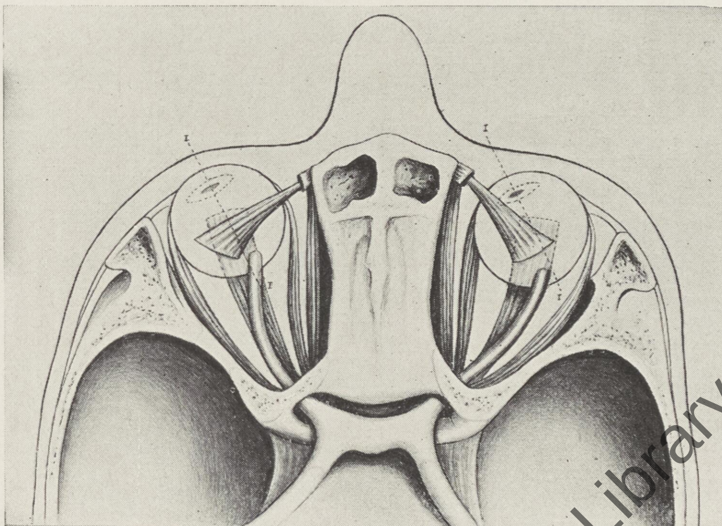


FIG. 234.—LOOKING DOWN AND TO LEFT.

The twelve muscles must function harmoniously in every movement of the two eyes in order to have comfortable binocular single vision. This means that both eyes have an image of the same object simultaneously. Binocular single vision is perfected when the two images are fused into one. This affords stereoscopic vision with an appreciation for the details of "relief" and the third dimension of an object, as well as an estimate of its distance.

The cardinal directions of gaze or rotation are "out," "in," "up and out," "up and in," "down and out," and "down and in." It will be seen that each eye employs the same muscles differently to perform the several conjugate rotations. In the following table the student will avoid confusion if he will orient his position as though he were standing behind his own eyes. The directions indicated will then correspond to his own imagined eye movements. The cardinal rotations are given.

Looking to the Left (see Fig. 232)

LEFT EYE	RIGHT EYE
Lateral rectus assisted by the obliques.	Medial rectus assisted by the superior and inferior recti.

(Transpose for *Looking to the Right,* see Fig. 233.)

Looking Up and to the Right (see Fig. 233)

LEFT EYE	RIGHT EYE
Medial rectus, inferior oblique; the superior rectus intorts.	Lateral rectus, superior rectus; the inferior oblique extorts.

Looking Down and to the Right

LEFT EYE	RIGHT EYE
Medial rectus, superior oblique; the inferior rectus extorts.	Lateral rectus, inferior rectus; the superior oblique intorts.

Looking Up and to the Left

LEFT EYE	RIGHT EYE
Lateral rectus, superior rectus; the inferior oblique extorts.	Medial rectus, inferior oblique; the superior rectus intorts.

Looking Down and to the Left (see Fig. 234)

LEFT EYE	RIGHT EYE
Lateral rectus, inferior rectus; the superior oblique intorts.	Medial rectus, superior oblique; the inferior rectus extorts.

Looking Up	*Looking Down*
EACH EYE	EACH EYE
Superior rectus, inferior oblique.	Inferior rectus, superior oblique.

THE NEUROLOGY OF OCULAR MOVEMENTS

The lateral rectus is supplied by the abducens, the superior oblique by the trochlear, and the remaining muscles are supplied by the motor oculi nerve. Pupillary contraction, accommodation and convergence are governed from the nucleus of the motor oculi.

Cortical innervation comes from the frontal and occipital lobes (Fig. 235). The frontal center is situated at the foot of the second frontal convolution in the motor area near the center for movements of the head. It presides over voluntary conjugate eye movements. The occipital center is situated in or near the visual cortex. Visual sensations reach this center and stimulate the frontal center for voluntary rotations, or the basal nuclei for involuntary or automatic rotations.

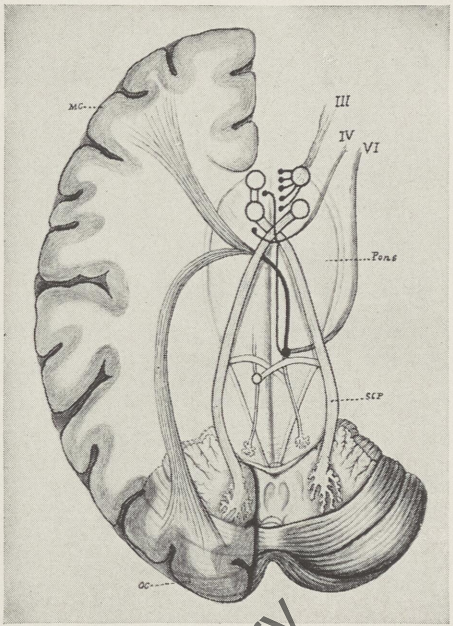


Fig. 235.—Rotation Centers, Schematic.

MC, motor cortex, and *OC*, visuomotor area, with pathways to abducens nucleus. *SCP*, superior cerebellar peduncle; *Pons*, pons; III, IV and VI, motor oculi, trochlear and abducens nerves traced from their nuclei.

The nucleus of the abducens nerve includes a pontine nucleus or is intimately associated with one. Fibers proceed from cells in the abducens nucleus along the medial longitudinal bundle to the cells in the opposite motor oculi nucleus that supplies the medial rectus of that side (see Fig. 235). An impulse to turn the eyes to the right stimulates the right lateral rectus and the left medial rectus, so that both muscles work in harmony for precise binocular adjustment in the new position of the visual lines.

The so-called "fusion area" of the retina might also be called the attention area. The term "attention-fixation" area is appropriate, because when the image of an object falls in this area it excites the attention, and attention generates a visuomotor impulse for fixation. Natural fixation is binocular or bifoveal, and the result is either simultaneous macular perception or fusion. Consequently, the strongest impulse for fusion occurs when the image of an object falls in the attention area. In simultaneous macular perception the two images are superimposed, while in fusion they are blended into a single image.

The superior colliculi are important organs to coördinate movements for bifoveal fixation. The eyes are directed toward any object that attracts attention; they retain fixation on a moving object, with or without head rotations. Impulses pass through the cerebellum before they reach the colliculi.

The cerebellum is essentially a nucleus for attaining and maintaining muscular equilibrium; its function is to control the quantity of impulse that goes to each muscle in a given motion. It governs the direction and extent of ocular

rotations; by proprioceptive muscular sensations the individual is enabled to determine the directions of objects about him, and their relation to his position and to each other. It is the important organ in orientation.

Visual orientation is objective or subjective. Objective refers to the ability to determine the relative position of objects in space to each other; subjective refers to the ability of the individual to determine his position relative to the situation of objects about him.

Fixation is acquired. Rays of light from an object enter the eye and impinge on the retina. An impulse to identify or examine the object causes the eye to move about until it secures the clearest image of the object. The fovea is the area of sharpest perception, and each eye searches until it secures foveal fixation. Experience and training are necessary to accuracy in locating objects. Bifoveal fixation is cultivated and becomes a habit.

Images are projected along the paths of the light rays that have entered the eye from objects, and by experience the images are projected so that they occupy the positions of the objects from which the rays originated. An object situated to the right forms an image on the left half of the retina and is projected into the right field; an image formed on the right half of the retina is projected into the left field (see Fig. 122).

Every percipient part of the retina has some capacity for image formation and projection. An image from any certain area of the retina is habitually projected into a definite position in the visual field. When both eyes are fixed on an object it forms an image at a certain point on the right retina, and at a certain point on the left. These are *corresponding points*. All other points are disparate.

Images formed on corresponding points are projected and either superimposed or *fused*. Fusion is the essence of binocular single vision. The images seen with the two eyes are not strictly identical, because the eyes are separated by a little distance. The right eye sees more of the right side of an object, and the left eye sees more of the left side. A box or block having two equal sides joined at a right angle is held before the eyes so that the two sides are seen equally well. When the left eye is closed the right side of the box seems broader, and when the right eye is closed the left side seems broader.

Images formed on disparate points are projected and are seen as two objects. Fixation is directed toward a distant object; a pencil held at arm's length between the eyes and the object of fixation is seen double. When fixation is directed toward the pencil, the distant object is seen double. This is disparation: the eyes are directed toward two objects which are unequally distant.

The *line of sight* or axis of vision extends from the fovea to the object. When fixing on the distant object, its image is formed on the fovea of each eye, and the image of the pencil is formed on the temporal side of each fovea; if the right eye is closed the left pencil disappears, and if the left eye is closed the right one disappears.

When fixing on the pencil, its image is formed on the fovea of each eye, and the image of the distant object is formed on the nasal side of each fovea; when the right eye is closed the right distant object disappears, and when the left eye is closed the left one disappears. Images formed on the temporal side of the fovea are projected into the nasal field, and images formed on the nasal side are projected into the temporal field (Fig. 236).

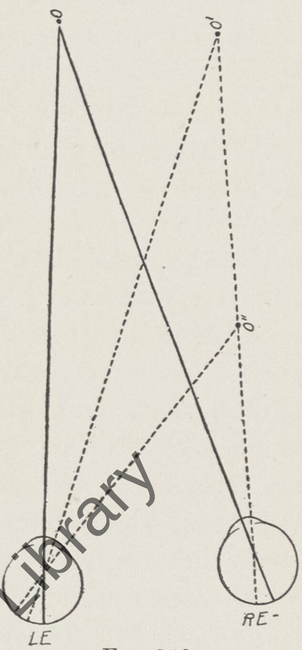


Fig. 236.

When an image is formed on the fovea of one eye, and on a point outside the fovea of the other eye, two images are projected; one is projected truly and the other falsely. This constitutes double vision or diplopia. Each retina projects the image that belongs to it into its accustomed place in the binocular field of vision. The *line of fixation* extends from the center of rotation to the object of fixation. It is the function of the extra-ocular muscles to place the lines of fixation in such a position that the image of an object will fall on the fovea of each eye. When one line of fixation is inaccurately placed the projection is false, and the eye to which the false projection belongs is said to *deviate*.

Eyes may maintain fixation, and yet seem to deviate. The curvature of the cornea, if completed, would form an ellipse, the major axis of which is the corneal axis. This axis and the line of fixation form the *angle kappa,* which may resemble a deviation (see also angle gamma, page 316).

Deviations are of two kinds, manifest and latent. An eye that deviates so widely that no attempt is made to fuse the two images rarely causes painful symptoms. A latent deviation is discovered only by tests that dissociate the images of the two eyes. It is a *tendency* toward deviation which is corrected by muscle action, and the excessive impulses required to maintain fusion are responsible for the symptoms of headache, usually frontal, and asthenopia or painful sight. The recognition of anomalies of muscle action will clear the diagnosis in many obscure cases of functional nervous disease and neuroses.

MUSCLE PARALYSIS

Signs and Symptoms.—The identification of the kind and character of a deviation is necessary to its successful management. Deviations are usually due to paresis or to weakness of a single muscle; since these are readily visualized by tests, they will be considered first. One eye will sight the test object, and it is called the sighting, fixing or dominant eye; the other is called the deviating eye.

The patient is seated facing the light so that the positions and movements of the eyes can be observed. A small test object, such as a pin with a small head, is held about 40 centimeters (16 inches) directly in front of the eyes. A manifest deviation and its direction are evident to inspection. The object is carried to the right and left, and in each of the diagonal directions. The failure of one eye to follow the object, and the direction in which the failure occurred are noted.

The patient is then taken into the dark room and seated 6 meters (20 feet) from a test light which should be no higher than the eyes, but rather lower. The trial frame is adjusted, the distance correction is placed in the cells, and a red glass is placed before the right eye. Either the light or the patient's head must be stationary. In the tests the light is carried in the several desired directions, or the patient's head is rotated to obtain similar effects; in either case the eyes are constantly directed toward the light. For the near test, the light from a self-illuminated ophthalmoscope may be used. The object is to furnish dissimilar images for the two eyes.

Duane simplified the tests so that the interpretations are subject to little or no confusion; the *red* image always belongs to the *right* eye and the white image to the left eye. Fixation can be made with either eye indifferently. Where the images are side by side horizontally, the paralysis belongs to a lateral rotator. When the red image is on the right the diplopia is homonymous; when it is on the left the diplopia is heteronymous or crossed. In homonymous diplopia the visual axes converge; in crossed diplopia they diverge.

When the distance between the images increases on directing the eyes toward the right, the right lateral rectus or the left medial rectus is paretic; when it increases as the eyes are directed toward the left, the left lateral rectus or the right medial rectus is paretic. *The diplopia increases in the direction of the action of the paralyzed muscle.*

Where the images are separated vertically, one or more of the elevators or depressors are affected. When the red image is above, and the separation increases as the eyes are directed upward, a right elevator is affected. When the separation increases as the eyes are directed downward, a left depressor

is affected. When the white image is above, a left elevator or a right depressor is affected. Since there are two elevators and two depressors for each eye, the tests must be carried further in order to identify which muscle is affected.

Beginning with a vertical diplopia, the eyes are directed horizontally to the right and then toward the left. When the vertical separation of images increases as the eyes are turned toward the right and upward, the right superior rectus is affected when the red image is above, and the left inferior oblique is affected when the white image is above. A similar separation toward the left and upward means that the left superior rectus is affected when the white image is above, or that the right inferior oblique is affected when the red image is above.

When the separation increases as the eyes are turned toward the right and downward, the right inferior rectus is affected when the red image is below, and the left superior oblique is affected when the white image is below. A similar separation toward the left and downward means that the left inferior rectus is affected when the white image is below, or the right superior oblique is affected when the red image is below.

Frequently the discrimination can be made by horizontal rotations without any upward or downward movements of the eyes. The rules are exactly the same. The essential difference between paralysis of an elevator and a depressor, when the eyes are directed in the primary position, is that the red image is above in paralysis of a right elevator, and below in paralysis of a right depressor. The paralysis belongs to the muscle that would unite the images if it were active. It is unnecessary to consider the inclination of the image due to torsion.

Paralysis of individual muscles is often accompanied by characteristics which confirm the tests based on diplopia. In the following discussion the muscles of the right eye will be considered; that eye will be covered with a red glass, and its image will be alluded to as the red image. The left eye will fix the light, and its image will be white.

Lateral Rectus.—The right eye converges toward the left since the medial rectus is unopposed. Rays from the test light fall on the nasal side of the right retina, consequently the red image is projected to the right; the diplopia is homonymous. The patient is instructed to gaze intently at the light and note the distance between it and the red image; he is then instructed to look at the red image, and he will notice that the distance has increased. Since the red image is to the right of the white one, the patient must rotate his left eye to the right to fix the red image. The effect is the same as carrying the light to the right, or rotating the face to the left.

The left eye is occluded and without turning the head the patient is directed

to quickly point toward an object on his right. He will point to the right of the object. The interpretation for subjective localization in direction is gauged by the motor impulse sent to the right lateral rectus, which is excessive in the presence of paralysis. That impulse would ordinarily carry the line of fixation of the right eye far to the right of the object. A prism of the proper strength held base out before the right eye will superimpose the two images. To avoid diplopia, the patient may habitually turn his face to the right, or toward the paralyzed muscle.

Medial Rectus.—The right eye diverges from the left since the lateral rectus is unopposed. Rays from the test light pass through the red glass and fall on the temporal side of the right retina, consequently the red image is projected to the left; the diplopia is crossed.

The patient looks at the light and notes the distance between it and the projected red image. He then looks at the red image and notices that the distance is increased. The left eye is occluded and without turning the head the patient is told to quickly point toward an object on his left. He will point to the left of the object. A prism of the proper strength held base in before the right eye will superimpose the two images. To avoid diplopia, the patient may habitually turn his face to the left, or toward the paralyzed muscle.

To differentiate between a paralysis of the lateral rectus and one of the medial rectus it is only necessary to know that the diplopia is homonymous in the former and crossed in the latter. To differentiate between paralysis of a right or left rotating muscle it is only necessary to know that the distance between the images increases as the eyes are rotated in the direction of the paralyzed muscle, or in the direction that the normally acting muscle would rotate the eye.

If the vertically acting muscles could contract individually, each would rotate the cornea in a definite direction. Such rotations are inconsistent with what actually occurs in health, but it will be helpful to imagine these actions in the study of paralyses of individual muscles. The superior rectus, acting alone, would rotate the cornea up and in; when it is paralyzed the cornea rotates down and out. The inferior oblique would rotate the cornea up and out; when it is paralyzed the cornea rotates down and in. The inferior rectus would rotate the cornea down and in; when it is paralyzed the cornea rotates up and out. The superior oblique would rotate the cornea down and out; when it is paralyzed the cornea rotates up and in.

Superior Rectus.—The right eye deviates toward the right and slightly downward. Rays from the test light pass through the red glass and fall on the inferior temporal quadrant of the retina. The red image is projected upward and nasally. The diplopia is both crossed and vertical. As the eyes are directed

upward and to the right, the distance between the images increases. To avoid diplopia the patient may turn the face upward and to the right, and may incline the head toward the left shoulder. Ptosis is occasionally associated with paralysis of the superior rectus.

Inferior Oblique.—The eye deviates toward the left and slightly downward. Light rays fall on the inferior nasal quadrant of the retina and the red image is projected upward and temporally. The diplopia is both homonymous and vertical. As the eyes are directed upward and to the left the distance between the images increases. The patient may turn the face upward and to the left, and may incline the head toward the right shoulder.

The motor root of the ciliary ganglion is given off from the nerve to the inferior oblique. Paralyses of pupillary contraction and of accommodation are occasionally associated with paralysis of the inferior oblique.

Inferior Rectus.—The eye deviates toward the temple and slightly upward. The diplopia is crossed and vertical; the red image stands lower in the field than the white one. As the eyes are directed downward and to the right the distance between the images increases. The face may be turned downward and to the right, and the head may be inclined toward the right shoulder.

Superior Oblique.—The eye deviates toward the nose and upward. The diplopia is homonymous and vertical; the red image stands lower in the field than the white one. As the eyes are directed downward and to the left the distance between the images increases. The face may be turned downward and to the left, and the head may be inclined toward the left shoulder. Isolated superior oblique paralysis is rare. It has been observed following operations on the frontal sinus.

In paralysis of depressors the red image may seem nearer to the patient than the white image. This illusion is probably due to the habit of associating the lower visual field with objects that are actually nearer to the observer, or because the background for the image is nearer.

Any or all of the muscles that are innervated by the motor oculi may become paralyzed. The signs of this condition are ptosis, elevation of the eyebrow, slight proptosis, dilated pupil, paralysis of accommodation, deviation of the affected eye toward the temple and slightly downward, face turned toward the eye affected, and the head inclined backward. There is limitation of motion nasally and upward. The diplopia is crossed. The distance between the images increases on looking nasally, upward or downward. In looking up, the red image is higher, and in looking down it is lower. Bilateral cases are difficult; symmetrical ptosis, pupillary dilatation, paralysis of accommodation, and the inability to look upward or nasally may help in the diagnosis.

Objective Signs.—One eye deviates, especially when an attempt is made to

look in the direction toward which the paralyzed muscle would normally rotate the cornea; the *excursion* of the affected eye is *limited* in that direction. The patient who has diplopia may elect to turn the face to the right or left, and to incline the head toward one shoulder or the other to avoid the effects of the double images. The face may be raised in paralysis of an elevator, or lowered in paralysis of a depressor.

Ocular torticollis is a name applied to an expression of these paralyses. The head is inclined toward one shoulder, and the face is turned to one side and elevated or depressed. To differentiate this variety of torticollis from others of different etiology, it is only necessary to cover the affected eye; the false image is excluded and the necessity for the awkward position is removed. The sign may not disappear by occluding the eye that is not paralyzed. This kind of torticollis is not painful, except from fatigue due to constantly holding the head in an unusual and strained position.

Subjective Symptoms.—The patient has false projection and diplopia. This confusion interferes with the muscle sense and tends to produce vertigo. The vertigo may be differentiated from other varieties by occluding the affected eye. It may not disappear by occluding the eye that is not paralyzed.

The signs and symptoms are modified in various ways. There are variations in the kind and degree of paralyses, according to the number of muscles involved, their functions, and the character of the etiological factor. Differences may exist between the manifestations in distant and in near vision. The age of the lesion usually brings about changes in the symptoms, since the patient learns to correct misinterpretations of his muscle sense, or acquires the habit of suppressing the image that belongs to the deviating eye. The progressive contraction of the opponent of the weakened muscle often confuses the diagnosis.

Diagnosis is generally made on the presence of three principal manifestations, and the signs and symptoms that depend upon them. These are deviation of the visual axis, restricted rotations, and diplopia.

An apparent paralysis of the lateral rectus may exist when actually the medial rectus of the same eye is spastic. The eye will not rotate temporalward with both eyes open, but when the sound eye is occluded the affected eye will follow an object as it is carried into the field of action of the apparently paralyzed muscle.

The screen or cover test is employed to detect slight degrees of paralysis or weakness. A suitable object of fixation is held about 40 centimeters (16 inches) in front of the eyes. The patient is directed to fix on the test object. One eye at a time is alternately covered and uncovered with an opaque card. Should the right eye deviate when covered, and fix along with the left when uncovered, the deviation is latent only. Should the right eye deviate regard-

less of being covered or uncovered, the deviation is manifest and the eye squints. Should the right eye deviate only when covered, it may be noticed that the left fixes when the right is covered, and deviates when the right is uncovered; in that case the left eye squints. The left eye is tested in the same way. The test should be repeated a number of times. In alternating squint the right eye will deviate sometimes, and at others the left will deviate.

Secondary deviation is to be compared to primary deviation. Primary deviation belongs to the affected eye whether its muscles are paretic or spastic. The deviation of the suspected eye is estimated *while under cover*. The cover is then transferred to the supposedly sound eye and its deviation is estimated. In cases of paralyses the secondary deviation is greater than the primary; in spastic conditions the primary deviation is greater than the secondary. The deviations are equal in comitant * squint.

The amount or extent of primary deviation can be determined by the employment of prisms and the covering card. Prisms of increasing strengths are placed before the deviating eye, which is covered *after* placing each prism. The apex of the prism must be placed in the direction of the deviation. When the eye is covered by the correct prism it should not move behind the card. The amount of secondary deviation is determined similarly.

The amount of deviation may be estimated on the perimeter. The deviating eye is placed before the perimeter in the position for taking its visual fields. The point of fixation should be 6 meters (20 feet) distant in line with the defective eye and the zero mark on the arc of the perimeter. The patient is directed to observe the point of fixation with the sound eye.

A light, such as is available in the self-illuminated ophthalmoscope, is carried along the arc of the perimeter. The light will be reflected from the cornea. When the reflection coincides with the center of the pupil, the position of the light along the arc of the perimeter is read off in degrees as indicated on the scale (Fig. 237). The examiner's eye should be in line with the light and the observed pupil.

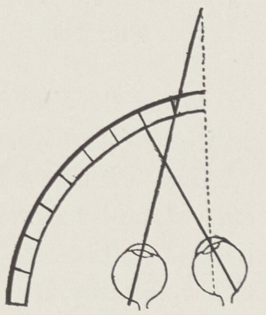


Fig. 237.

The amount of the deviation for distance can be measured by the strength of the prism required to unite the double images. Cases of combined vertical and horizontal diplopia can be measured by one prism for the vertical and another for the horizontal deviation.

Where one eye suppresses the image belonging to it, it is necessary to devise a plan by which the suppressing eye may be made conscious of an image; the images of the two eyes must be dissociated. For this purpose one eye is

* Author's choice of terminology in preference to concomitant.

covered with a red glass, a Maddox rod, or a Maddox double prism. The red glass only serves to dissociate colors, the rod produces a different image from that seen by the uncovered eye, and the double prism gives two images to the eye behind it.

Maddox rods are single or multiple, uncolored and colored. The single rod is a strong cylinder, and the multiple rod consists of several such cylinders mounted parallel in one plane for use in the trial frame (Fig. 238). When looking at a light through a cylinder, the image appears as a line at a right angle to the axis of the cylinder. The patient should wear an approximate correction for any existing visual error.

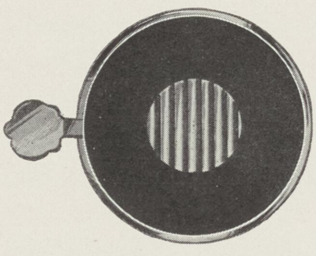


Fig. 238.—Multiple Maddox Rod.

The rod is placed vertically before the right eye; the linear image is horizontal. When the line is seen above or below the light (see Fig. 239, *B* and *C*), the visual axis of one eye deviates upward or downward. In vertical deviations the higher looking eye is designated. The strength of the prism, placed base down before the higher eye, that is required to bring the images to the same horizontal level (see Fig. 239, *A*) measures the amount of upward deviation.

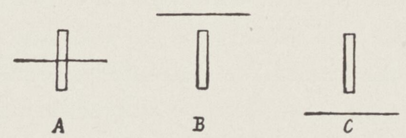


Fig. 239.

The rod is rotated 90 degrees so that the line will be vertical. When the line appears on the right side of the light (see Fig. 240, *B*) the images are homonymous. When the line is seen on the left side of the light (see Fig. 240, *C*) the images are crossed.

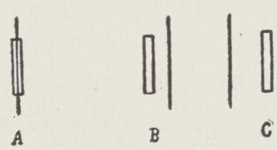


Fig. 240.

When the line passes through the light in both the vertical and horizontal positions (see Figs. 239, *A* and 240, *A*) the bifoveal fixation sense is strong.

The test serves to detect the presence of weak muscles as well as the presence of faulty motor impulses. With a varying relation in which the line may be first on one side of the light, then through it, and again on the other side, either the impulse for fixation or motor innervation may be at fault (see Fig. 235), or the muscles may be weak. In multiple sclerosis the patient cannot maintain definite positions of the light and the line; such a case represents defects in the central nervous system. Myasthenia gravis, with its constantly varying muscle tone, represents an example of weakness.

The Maddox double prism consists of two prisms set base to base in a rim for use in the trial frame. When this is properly adjusted before the eye, a

distant light appears as two lights (Fig. 241). When the bases are set horizontally the images are one above the other; when set vertically they are side by side. When the uncovered eye also fixes the light, three images of it will be seen: Two by the covered and one by the uncovered eye. The device is used in the same manner as the Maddox rod. A red glass placed before the uncovered eye assists the patient to describe to the examiner the relative positions of the three lights as he sees them.

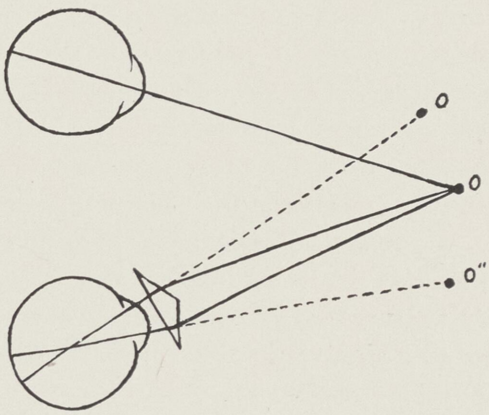


Fig. 241.—Triplopia with Double Prism.

Occasionally the physician meets a patient who has total monocular suppression while both eyes are open and directed toward the subject; the eye with the weaker vision suppresses as long as the other eye sees the object. It is not amblyopic, since it will take up fixation when the other eye is covered, and its form fields are not contracted. No stratagem seems to be clever enough to beguile the weaker eye to recognize the image belonging to it while the other eye fixes.

Diplopia is overcome when the patient learns to suppress or ignore the image from one eye. Where the paralysis affects the third nerve, an accompanying ptosis will occlude the pupil of the deviating eye.

Prognosis.—This depends upon the cause and nature of the lesion. Peripheral paralyses are often transient. Nuclear lesions may be remediable. Permanent paralyses are usually succeeded by progressive contractions of opposing muscles; these draw the visual axis so far to one side that the image belonging to the affected eye ceases to bother the patient. Habitual contraction of the neck muscles may occur in paralyses of elevators or depressors.

Etiology.—The causes of paralyses of the extra-ocular muscles are found within the orbit or within the cranial cavity. Aside from congenital structural defects and anomalies, paralyses of orbital muscles generally constitute expressions of disease, rather than disease of the affected muscles.

Orbital lesions for the most part act mechanically. A wound may injure or sever a muscle or its nerve, and so produce the effect of a paralysis. In general, lesions within the orbit crowd or rotate the globe to one side, and produce a deviation of the visual axis with fixation of the globe and a simulation of paralysis. In paralysis, passive motion meets no resistance and causes no pain. Usually in orbital disease passive motion causes pain or meets resistance. Proptosis is frequently present in orbital lesions.

Paralyses due to intracranial lesions are classed according to the extent of the consequences, and according to the location of the lesion. Ophthalmoplegia externa refers to a paralysis of one or more extra-ocular muscles; ophthalmoplegia interna is paralysis of the iris and ciliary muscle; ophthalmoplegia totalis refers to the coexistence of the external and internal forms. One or both eyes may be affected. The seat of the lesion is infranuclear or between the nucleus of the nerve and the muscle, nuclear or in the nucleus, and supranuclear or between the nucleus and the motor centers.

Infranuclear lesions can be unilateral or bilateral, and they can affect one muscle or several. *Nuclear* lesions usually produce bilateral symptoms which are not necessarily equal for the two sides. *Supranuclear* lesions do not affect muscles singly; the usual manifestation is an inability to perform one or more conjugate movements.

Paralyses are caused by hemorrhages, exudates, inflammations, degenerations, acute infectious diseases, foci of infection in the nasal sinuses, nose, throat and ear, colds, intestinal torpor, toxemias, various diseases of the brain and meninges, poisoning, epidemic encephalitis, syphilis, tabes, general paralysis, gummata, tumors and basal fractures. Ordinarily the prognosis is good in acute conditions, but in chronic cases the paralyses are apt to be permanent.

Vascular lesions comprise arteriosclerosis, arterial and arteriovenous aneurysm and rupture or compression of vessel walls. Hemorrhages and thromboses act like tumors that produce pressure; the onset of paralysis is rapid and sometimes abrupt. Thrombosis of the cavernous sinus is practically always accompanied by paralysis of one or more ocular muscles. This is due to the intimate relationship of the nerves to the sinus. The abducens passes through the sinus; the trochlear and motor oculi are embedded in its dural wall.

The abducens nerve is peculiarly prone to injury because of its exposed position between the pons and the dorsum sella of the sphenoid bone, its long course within dural layers which are intimately adherent to the underlying bone, and its relationships as it passes between the tip of the petrous portion of the temporal bone and the posterior clinoid process. Inflammatory exudates which bathe it in the first portion, and meningitides of the base which compress it in the second and third portions produce abducens paralyses.

Paralysis of the abducens is often one of the early signs of syphilis in the nervous system. A basal meningitis may be a factor in causing it.

The petrous portion of the temporal bone is often quite cellular, and purulent processes from otic inflammations occasionally extend as far as the tip of the bone and involve the dural tissue at the point where the nerve crosses the tip. Gradenigo's syndrome of otitis media, abducens paralysis and headache, all on the same side, illustrates associated processes in this locality.

Basal fractures which extend as far as the tip of the petrous bone sometimes cause paralysis of the abducens.

The trochlear nerves are occasionally caught at this point also. The course of these nerves from the dorsal surfaces of the cerebral peduncles serves to protect them to a considerable extent, or until they arrive at the point of crossing over the bony notch. A few cases of bilateral trochlear paralysis have been reported in connection with tumors of the pineal gland.

Basal meningitis often causes a partial paralysis of the motor oculi nerve; some of the fibers become involved, but not necessarily all of them. It is usually unilateral. The effect is more apt to be bilateral when due to syphilis. Tumors of the hypophysis may cause paralysis.

Lesions in the cerebral peduncles generally involve all muscles supplied by the motor oculi, including the iris and ciliary body. When the muscles of the face and tongue are also involved, it is called Weber's paralysis. This is seldom bilateral. Sometimes the lesion includes the red nucleus, in which case the body, arm and leg on the opposite side are subject to tremors; Benedict's syndrome.

In locomotor ataxia, general paralysis of the insane and cerebral syphilis, the paralysis is occasionally limited to the motor oculi or to some of its branches. In the first disease it is often found in the preataxic stage; it may disappear, but it will recur. The tissues affected in these diseases are believed to be the gray matter about the cerebral aqueduct and the peduncles.

Lesions in the cerebellar peduncles have been known to cause Magendie's squint or skew-deviation. One eye looks up and out while the other looks down and in, or one may look up and in while the other looks down and out. Acoustic nerve tumors and new growths in the cerebellopontine angle are generally accompanied by abducens paresis and deafness on the same side, vertigo, and signs of increased intracranial pressure.

In nuclear paralysis the diplopia usually appears very early. The motor oculi is prone to injury, although the paralysis is apt to be but partial; there is generally an associated ptosis. The facial nerve is often affected with the abducens. Hemorrhages into the nuclei are accompanied by an abrupt paralysis; other motor disturbances may be present. Lethargic encephalitis often causes nuclear paralysis.

All supranuclear lesions occur in the motor cortex or in the tracts which connect the cortex with the nuclei, including the internal capsule. They may be destructive or irritative; the former causes paresis, and the latter contractures. Conjugate deviations of the eyes, or of the head and eyes, are expressive of this type of motor disturbance. Cortical innervation for the abducens comes from the opposite cerebral hemisphere; the supranuclear tracts decussate in the pons.

A destructive lesion on the right side above the point of crossing affects the nucleus of the opposite or left side. The left lateral rectus can receive no voluntary motor impulse, consequently the left eye cannot be rotated toward the left. The impulse cannot reach the right medial rectus, and the right eye cannot be rotated toward the left, although the power of convergence is retained because that center is not affected. Conjugate rotation to the left is suspended. The eyes deviate to the right, or toward the side of the lesion; the patient looks toward the lesion.

Should the lesion occur further down in the pons but still on the right side, it will affect the supranuclear tract from the left hemisphere to the right abducens nucleus. Neither eye can be rotated toward the right side, and both will deviate to the left (see Fig. 232) or away from the lesion, although the lesion is on the same side as before. Where the lesion is in the abducens nucleus, the eyes cannot be rotated toward that side, and they will deviate to the opposite side.

An irritative lesion produces expressions which are opposite to those that have been described. Such a lesion in the higher position causes the eyes to deviate to the left; when in the lower position they deviate to the right. Other symptoms will be present with such lesions.

Conjugate deviations are often associated with either hemiplegia or contractures of the limbs on one side. When a destructive lesion occurs in the higher position in the pons, the eyes look toward the side of the lesion and away from the paralyzed arms and legs. When it is located in the lower position, the eyes look away from the lesion and toward the paralyzed limbs. An irritative lesion in the higher position causes the eyes to look away from the lesion and toward the spastic side. When it is in the lower position the eyes look toward the lesion and away from the contracted limbs. These observations are based on lesions located on the right side; the left limbs will be affected (see Fig. 235).

Influenza and diphtheria cause paralyses of eye muscles; they are more apt to paralyze accommodation. Botulism and ptomain poisoning act on the intrinsic muscles, although they may cause some paralyses of extrinsic muscles. Lead may act on either set. The prognosis in alcoholic paralyses should be guarded; many cases do not recover use of the muscles.

Spasticity.—An eye may deviate in consequence of a spasm of one or more muscles as readily as from paralyses of opposing muscles. As in paralyses, the amount of the deviation varies in different directions of gaze. It is sometimes difficult to differentiate between paretic and spastic deviations. The following points are of value.

The deviation in spasm is greater when the healthy eye fixes; the primary

deviation is greater than the secondary. When the spastic eye fixes, the visual axis of the sound eye approximates to that of the fixing eye, and deviates but little. When rotation is performed in the direction of the action of the spastic muscle, the excursion is excessive, and the cornea of that eye moves faster and further than that of the sound eye.

Diplopia exists, and the distance between the images increases as the eyes are turned in the direction which corresponds to the field of action of the spastic muscle, because that muscle overacts. Should the distance between homonymous images increase as the eyes are turned toward the right, the right lateral rectus is paretic or the left medial rectus is spastic. In the one case the right eye cannot be rotated fully toward the right, while in the other the left eye is rotated too far toward the right.

Projection is weak in the presence of spasticity. The sound right eye is occluded, and the patient is directed to point toward an object on his right. He will not point to the right far enough. The motor impulse required to turn his left eye to the right is weak, and guided by the strength of that impulse the distance is underestimated.

Objects in the visual field may appear to move, and thus cause vertigo.

Spasm may be due to extra power in the muscle as compared with its weak opponent, and this power may arise partly from an insertion abnormally near the cornea. The majority of spasmodic movements occur in convergence and in divergence. The opponent of a paralyzed muscle may become chronically spastic. Cerebral irritation usually affects both eyes, so that the deviations are conjugate. Irritative pontine lesions also may cause spastic conjugate deviations.

STRABISMUS OR SQUINT

Classification.—*Orthophoria* is a term used to express the idea that bifoveal fixation is habitually comfortable. *Heterophoria* means that bifoveal fixation can be maintained, but only with excessive motor impulses. *Heterotropia* means that one eye habitually deviates, and that bifoveal fixation does not exist.

The deviation or squint may be lateral, vertical or mixed. A lateral squint is convergent or divergent. In vertical squint one eye is directed higher than the other. In the mixed form the deviation has a diagonal direction.

Squints are clinically classified according to behavior. A *monocular* squint affects one eye only; an *alternating* affects either eye indifferently. A *constant* squint is present at all times; an *intermittent* one is present only a part of the time. A *continuous* squint is present for all distances; a *periodic* is present in either near or distant vision but not in both. In *comitant* or *concomitant*

squint the angle of deviation is constant in all positions of the eyes; in *non-comitant* or paralytic, the angle varies in different positions and may be absent in some.

A monocular squint is due to a defect in the eye whereby bifoveal fixation is difficult or impossible. The causes have been ascribed to hyperopia, astigmatism, anisometropia, amblyopia, opacities of the cornea or lens, defects in the retina or choroid, central scotomata, defects in the optic pathways, muscle weakness, acute illnesses and so on. An alternating squint permits fixation with either eye, because the visual abilities of the two eyes are approximately equal. A peculiar type of alternating squint exists when the left half of a line of print is read with the right eye and the right half with the left eye. These eyes do not seem to rotate temporally.

A constant squint is due to some cause which is always operative. An intermittent squint is usually due to ocular or general fatigue which is not constantly present.

A continuous squint depends upon some cause which is always present. Thus a monocular squint may be also constant and continuous.

A periodic squint exists in two forms. It is said to be *directly* periodic when the deviation is present in near vision but not in distant, or when it is greater in near than in distant vision. The squint is *inversely* periodic when the deviation is present in distant but not in near vision, or when it is greater in distant than in near. Periodic squints must not be confused with the alternating type, in which distance is not a factor.

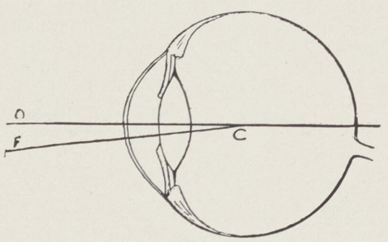


Fig. 242.—*FCO*, Angle Gamma.

An apparent squint sometimes exists. The *optic axis* passes from the center of the retina through the center of the lens. The *line of fixation* connects the center of rotation with the object fixed upon. The angle formed by the optic axis and the line of fixation is the angle *gamma*, which may be wide enough to resemble a squint (Fig. 242).

Etiology of Comitant Squint.—1. A physiological relationship exists between accommodation and convergence. Hyperopic eyes must accommodate for all distances. This stimulates convergence excessively, and in the effort to maintain binocular single vision the eyes converge so much that neither obtains clear macular perception. The patient learns to improve vision by fixing with one eye and allowing the other to deviate.

Myopic eyes must converge even for their far-points, and out of proportion to the amount of accommodation required. Neither eye obtains clear macular perception. Again one eye fixes while the other deviates.

This theory is incomplete, since the great majority of hyperopes and myopes do not squint, while some emmetropes do.

2. Anisometropia, or unequal vision in the two eyes, is found in some cases of squint. This may cause periodic deviation where one eye is used for distance and the other for near vision. Yet anisometropia can exist without squint.

3. Disturbances of motor innervation, in which there is an excess or an insufficiency of convergence or of divergence, cause some squints. It is probably but one factor, an inability to maintain bifoveal fixation.

4. Fusion is a faculty that is usually established not later than at six years of age. When the fusion sense has failed to develop, binocular single vision depends upon motor coördination. A protracted disturbance in coordination causes permanent squint. While there are some apparent exceptions, lack or loss of fusion accounts for more cases of squint than other factors do.

According to Landolt, a primary lesion in the centers for convergence and divergence can cause squint independently of such other factors as refraction, visual acuity and binocular vision. The diagnosis is based on the following characteristics: Secondary deviations and false projection are absent, diplopia is always present, and the distance between the images increases in looking either to the right or left. In paralytic squint the distance increases when looking in one direction, and decreases when looking in the other. In comitant squint diplopia is usually absent.

The patient who has comitant squint is rarely troubled with diplopia, because the image received by the deviating eye falls on a portion of the retina which is less sensitive than the macula, and is suppressed or ignored. In long-standing cases a false macula may be formed at the point where the image is received, and this pseudomacula enables the eye to employ eccentric fixation. The habitually fixing eye is occluded, and the patient is directed to touch some small object in front of him. This is accomplished with surprising celerity and accuracy, although the eye does not appear to be directed toward the object touched.

About 75 per cent of squints develop before the end of the sixth year. An arrest in the visual development of one eye leads to amblyopia. The power of fixation of an eye in a young child will be lost after about six months of nonuse. What is called "amblyopia ex anopsia" is often due to a reduction in vision which leads to a squint, although in some cases a squint is responsible for the deterioration of vision. Amblyopia may be congenital, with or without deviation.

Signs.—A manifest deviation is present. In performing rotations the eye with a convergent squint does not turn outward, while that with a divergent squint does not turn inward to the full extent. In convergent squint the eye

deviates in. Visual acuity is usually low from hyperopia. The pupil is often small. In divergent squint the eye deviates out. The conjunctiva is generally hyperemic and the lid borders are often swollen.

Symptoms.—There are subjective sensations of headache, eye pain, burning or itching of the eyelids, feeling of weight in the lids, and asthenopia from excessive motor impulses. Print is blurred, and the effort to read brings on general fatigue and drowsiness. Diplopia, nausea and vertigo may be present.

Converging Squint.—The visual axes must intersect at the object of fixation for clear binocular single vision. These axes are regarded as parallel for all distances of 6 meters (20 feet) or more. The most remote distance at which a point can be seen single by binocular vision is called the "far-point of convergence."

The "near-point of convergence" is the nearest distance at which a point can be seen single by binocular vision. The distance is measured by placing the end of a Prince's rule against one side of the nose 11.5 millimeters in front of the cornea, or against a trial frame adjusted at the proper distance from the eyes; a small object is made to approach the nose in the midline until it is seen double. The figure is read off in millimeters on the rule.

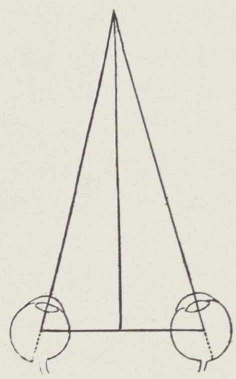


Fig. 243.—Meter Angle.

The meter angle or M.A. is the unit of convergence. This angle is computed from a base line which connects the centers of rotation of the two eyes (Fig. 243). The length of the base line is equal to the distance between the right, or left, borders of the two pupils (Fig. 244). The center of rotation lies about 13.5 millimeters behind the apex of the cornea, and the near-point is measured from about 11.5 millimeters in front of it; then 25 millimeters must be added to the figure read from the rule.

The standard unit, 1, is situated 1 meter in front of the mid-point of the base line, and it represents the convergence angle required for bifoveal fixation at that distance. At 2 meters (80 inches) the angle is half as much, or 0.5 M.A.; at $\frac{1}{2}$ meter (20 inches) the angle is twice as much or 2 M.A., and so on. To find the meter angle, divide 1,000 by the near-point in millimeters plus 25.

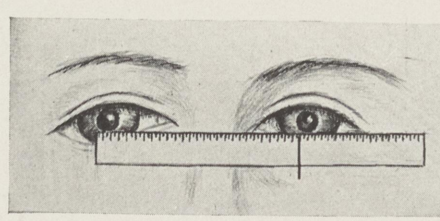


Fig. 244.—Measuring Interpupillary Width.

The average normal person should converge to as close as 35 millimeters from 11.5 millimeters in front of the

cornea, or about 60 millimeters from the base line. That is equivalent to about 16 M.A., or $1{,}000 \div (35 + 25)$. The meter angle and accommodation bear the same relation to distance.

The convergence impulse affects each medial rectus muscle equally. Motor coördination requires that convergence stimulation shall be accompanied by divergence inhibition. The balance between the medial and lateral recti is disturbed when one group gains or loses power in a measure not shared by the other.

When fixation is directed toward an object situated at infinity, there is no direct stimulation to convergence. But where the individual is hyperopic, he must employ his accommodation to see clearly. The employment of accommodation stimulates convergence, and this constitutes a state of esophoria for that distance. This is called the "accommodative type." A nonaccommodative type is found in some neuropsychiatric or debilitated patients. These illustrate convergence excess.

A myopic individual sees objects inside infinity distinctly, and his convergence is not adequately stimulated in the absence of accommodation. He therefore has convergence insufficiency.

The following table outlines the results of tests by means of which the varieties of convergence and divergence anomalies may be studied and differentiated.

Table of Excess and Insufficiency *

	Convergence Excess	Divergence Insufficiency	Mixed Forms
Esophoria	Greater for near than for distance	Greater for distance than for near	Present in both near and distance
Convergence near-point	Excessively close	Absent or negative	Normal or close
Prism convergence	Normal	Low or normal	Excessive or normal
Prism divergence	Normal	Low or negative	Low, absent or negative
	Convergence Insufficiency	**Divergence Excess**	**Mixed Forms**
Exophoria	Greater for near than for distance	Greater for distance than for near	Present in both near and distance
Convergence near-point	Remote; more than 90 millimeters	Normal	Remote or lost
Prism convergence	Low; difficult	Normal	Low or negative
Prism divergence	Normal	Excessive	Excessive

* Modified from Duane in Fuchs.

"Excess" refers to an active process in which the muscles are too strong, while "insufficiency" refers to a passive process in which the muscles are weak.

Convergence excess is readily confused with divergence insufficiency, and

one may lead to the other; in that case the forms are mixed. Long-standing cases of esophoria often lead to esotropia. Convergence insufficiency is easily confused with divergence excess, and one may lead to the other; when associated the forms are mixed. Protracted cases of exophoria may lead to exotropia. In all mixed cases one form was primary, and the characteristics of a mixed form will be influenced by the primary condition.

In the above table it will be noted that it is necessary to measure the extent of the lateral separation of the images for both distant and near fixation. Study of the table affords the helpful hint that esophoria and exophoria depend more upon disturbances of the function of convergence than of divergence.

Prism Divergence and Prism Convergence.—A red glass is placed before the right eye. A 1 or 2 D. prism is held base down before the right eye to produce vertical diplopia. As the *apex* is rotated toward the lateral or medial rectus the patient can see the images merge into one. Successively stronger prisms are used until the patient can no longer induce fusion. Convergence and divergence are binocular functions. The medial recti should overcome at least 6 D.; the lateral recti should overcome 1.5 D., and they rarely go beyond 4 D. By practice the muscles can greatly exceed these figures. The vertical rotators are tested by the amount of prism that they can overcome as a group.

The cover test is used to detect small amounts of heterophoria. A small object is held 40 centimeters (16 inches) in front of the eyes. The eyes are covered alternately with the card. In esophoria the eye behind the card deviates nasally, in exophoria temporally, and in hyperphoria it deviates vertically. As the card is repeatedly shifted from one eye to the other, the object appears to move with the card in exophoria, opposite the card in esophoria, and vertically in hyperphoria.

Hyperphoria.—This term signifies that the visual axis of one eye has a tendency to be directed higher than that of its fellow. It is applied to the higher looking eye by prefixing "right" or "left." Hyperphoria depends upon a weakness of depressors or an overaction of elevators. Headache, asthenopia and vertigo are common symptoms, and they are likely due to the strong motor impulses generated to correct the deviation and restore bifoveal fixation and fusion. The symptoms usually disappear after parallelism has been reëstablished by prisms or by muscle surgery.

These deviations often have highly latent characteristics, which is demonstrated by the *prolonged occlusion test.* The nonfixing eye is fitted with a pad which is left in place for a period of one day to one week, according to the examiner's judgment. The stimulus to bifoveal fixation relaxes. After wearing the pad for the prescribed time the patient is seated as for ordinary muscle testing. He is told to keep his eyes closed. The pad is removed. The trial

frames are adjusted and the patient's correcting lenses are placed in the cells; a Maddox rod is placed vertically before the eye that was occluded.

The patient is instructed to open both eyes, fix the light, close both eyes the instant he has located both images, and then announce his observation. Since the image belonging to the higher looking eye will be below, a prism is placed base down before that eye, or base up before the other eye. The process is repeated until a prism is found which places the images on a common horizontal plane. The tests are repeated for the reading distance and *position.* The patient must keep the eyes closed except during the momentary tests with different prisms.

Occasionally hyperphoria is associated with esophoria or exophoria. Then the condition is one of hyperesophoria or of hyperexophoria.

Hypertropia is a manifest vertical squint; the deviation will vary in different directions of gaze. Hyperphoria has little tendency to vary in amount.

Cyclophoria.—The vertical corneal meridian is rotated about the anteroposterior axis of the eyeball (see Fig. 231). It is caused by an excess or insufficiency of an oblique muscle. The behavior and significance of cyclophoria are not fully understood.

Asthenopia is an expression of the demand for oculomotor coördination when that function is fatigued or exhausted; it depends upon the inability to maintain binocular fixation without continuous effort. Astigmatism and hyperopia may or may not be the primary cause. When binocular fixation can be maintained with minimum motor impulses, pain is not present. When one image falls outside the attention-fixation area, there is no strong impulse for fusion; that image may be suppressed. The eye may or may not squint. Pain is not a symptom of suppression or squint.

Asthenopia may be modified by occupation. Outdoor work does not require the accurate binocular fixation demanded by such work as sewing, stenography and so on, especially when the lighting conditions are faulty. The highly sensitive individual is more prone to suffer from this cause than one who is less sensitive.

Treatment

Nonsurgical Treatment.—The earlier the treatment can be instituted, the better are the chances for successful management of the squint. While some cases improve with time and growth, they are exceptional. As a rule squint becomes progressively more marked. Since the majority of deviations are due to or are associated with errors of refraction, the first duty is to correct them.

Refraction should not be undertaken without a prolonged period of cyclo-

plegic preparation. Children under fourteen years of age should have atropin sulphate 0.5 to 2 per cent solution one drop in each eye three times a day for from three to five days. Scopolamin or hyoscin solutions, 0.1 per cent, can sometimes be advantageously substituted for atropin solutions, especially in older children. The faculty of accommodation should be suspended as completely as possible. The refraction is estimated by retinoscopy, and supplemented by test letter charts.

The prescription should correct all of the astigmatism. The spherical correction ordered will be calculated according to the state of the muscle balance. In convergence excess all hyperopia should be corrected; this will overcome the stimulating influence of accommodation on convergence in distant vision, and favorably modify that influence in near vision. In convergence insufficiency all myopia should be corrected, for it is desirable to create or increase the demand for accommodation to strengthen convergence. (See page 372, last lines.)

The patient or parents must be impressed with the necessity of wearing the glasses constantly. Very young children, even one year old, are easily taught to wear glasses, particularly when their vision is improved by lenses; the principal difficulty is to keep the spectacle frames properly aligned.

After having worn the correction for some weeks or months the tests ought to be repeated. Every change in the refraction or muscle balance calls for proper lenses to meet the new situation.

Cultivation of the Visual Functions.—Squint is frequently associated with poor vision in one eye. Vision and fixation can be substantially improved where the fovea can be developed. In the following exercises correcting lenses should be worn.

Exclusion of the better seeing eye can be accomplished with a bandage applied for periods of a quarter to one hour several times a day. This forces the child to use the weaker eye.

Atropin may be used in weak solutions in the better eye daily, or in stronger solutions once every week or two weeks, in order to compel use of the weaker eye. In such cases the weak eye must be used for near vision. When the visual acuity of the weak eye is very low the plan may fail, for neither eye can see well while the better one is under atropin. The child is taught to use the weaker eye by looking at or cutting out pictures, playing with small objects, and reading.

Binocular vision may be cultivated. Viewing pictures or special drawings through a stereoscope often helps the child to learn how to fuse the images he sees with the two eyes. The amblyoscope is employed for the same purpose, but it is tedious to use with very young patients. Neither of these instruments are of any certain value after the age of six years, but they should be tried. A

simpler exercise is "bar reading." A pencil is held upright between the eyes and the reading matter; in this position the pencil will conceal some letters from the right eye and some from the left, but none from both. As proficiency is attained, the pencil is held nearer and nearer to the printed page.

The recognition of diplopia is helpful when the patient habitually suppresses the image belonging to one eye and is unaware that he sees double. With a red glass over one eye and a green one over the other, the fixation light is seen as red with one eye and as green with the other. The dissimilarity attracts attention. With the idea of learning at what distance and in what position the lights are seen nearest together, the light is carried from 20 feet toward the patient, from the reading distance out to 20 feet away, to one side or the other, and up or down while the head is held immovable.

Once the patient is able to unite the images, he is encouraged to further efforts. Patience and perseverance will accomplish much. Should the patient be unable to fuse the lights in any position or at any distance, he can be aided by prisms. These are placed base out in esophoria, base in in exophoria, and base down before the higher looking eye in hyperphoria. The weakest prism with which the images can be united should be used at each practice period.

After the patient has acquired the ability to unite the images without the aid of prisms, the weak muscles can be further strengthened by the method which Maddox called "adverse" prism exercise. The prism is placed base in in esophoria and base out in exophoria. The patient is taught to overcome successively stronger prisms until he has normal prism convergence and prism divergence, and comfortable binocular fixation.

Adverse prism exercise is practiced in the following manner: With the light at the reading distance the prisms are placed base in for convergence excess, and base out for convergence insufficiency. With the light at 6 meters the bases are placed base in for divergence insufficiency, and base out for divergence excess. In mixed conditions the light is used at both distances, and the prisms are set as required.

Convergence can be strengthened by a simple exercise. A small-headed pin is held at arm's length directly in front of and below the horizontal plane of the eyes. Fixation is made on the pinhead, which is drawn *slowly* toward the nose. When the pinhead is seen blurred or doubled, the eyes are closed and the arm is extended. Where the approaching movement is not made slowly, one eye may suddenly deviate and simultaneously suppress the image of the object. The patient does not know what has happened, and the exercises do no good. The training should be done for periods of two minutes five or more times a day.

Convergence excess may be treated by instilling a drop of atropin in

1 : 30,000 to 1 : 15,000 solution into each eye on alternate days. The accommodation is relaxed and convergence is relaxed with it. Convergence insufficiency may be treated by instilling a drop of 1 : 300 to 1 : 150 solution of pilocarpin into each eye morning and noon. The pupil is contracted, and both accommodation and convergence seem to be stimulated.

Weak convergence has been strengthened by tincture of nux vomica, U.S.P., internally. Begin with twenty drops three times a day after eating. The amount is increased by one drop on each succeeding day for thirty days, when the dose will be fifty drops. The quantity is then decreased by one drop a day for thirty days, when the dose will be back to twenty drops. No doses may be omitted.

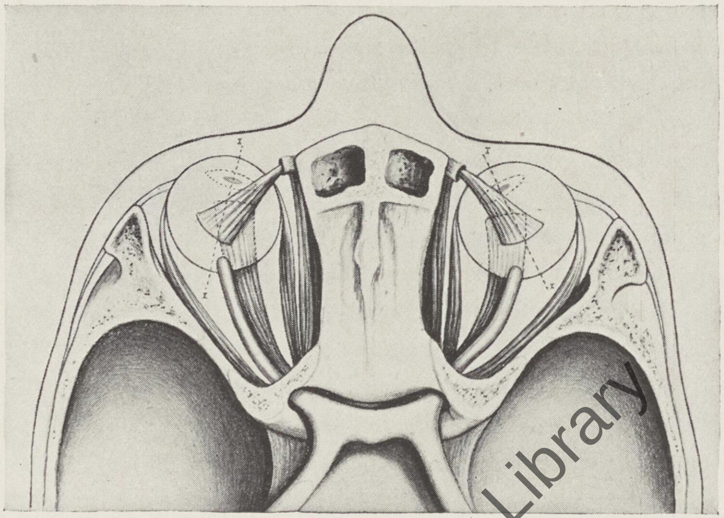


Fig. 245.—Convergence Downward, Reading Position.

Prisms are prescribed in comitant squints where the deviations cannot be corrected by muscle training. In applying a prism, the apex is placed in the direction of the squint. In lateral deviations divide the amount of prism and place half of it over each eye. The apices will be directed toward the nose in convergent squint, and toward the temples in divergent squint. The weakest prism with which the patient can obtain distant binocular fixation should be prescribed, and it is seldom advisable to exceed four centrads. Training should be continued while the prism is being worn, and its strength should be reduced when improvement permits.

It is good practice to prescribe prisms in comitant vertical deviations, but a half centrad should be deducted. For small and moderate deviations the prism is placed base down before the higher looking eye, except in paralytic *hypophoria*. Moderately high degrees may be corrected by dividing the prism

between the two eyes, base down before the higher looking eye and base up before the other. High deviations are not suitable for prism treatment; it is better to correct or partially correct by a muscle operation, and then give whatever prism is needed.

Prisms are without value in paralytic squints, since the amount of deviation changes as the eyes are rotated in various directions.

In the reading position, convergence is accompanied by the depressor action of the superior obliques (Fig. 245). It is possible for diplopia to exist in all positions of the eyes except this one, and conversely, it may exist in this position only. In the latter instance it is often the cause of drowsiness and inability to hold the attention when reading. This may account for the dullness of some children in school.

General and special conditions outside the eyes must receive attention. Obstructions and contact points in the nasal passages may be responsible for asthenopia. It is always advisable to explore this locality and the accessory nasal sinuses in all cases of squint, especially before resorting to operations.

Remote and reflex disturbances may be the cause of eye symptoms. The inability to maintain steady binocular fixation will suggest the possibility of myasthenia, unstable coördination, imbalance in the endocrine system, hysteria and neurasthenia.

Indications for Operation.—After other corrective methods have been used a reasonable time without effect, it is proper to consider surgical treatment. Such measures should be followed by the exercises already discussed. Fusion may become possible after operation where it was impossible before. Ordinarily, an operation will be unsuccessful unless glasses are worn. Operations carefully planned and skillfully performed, when preceded by thorough study and followed by appropriate treatment, will be successful.

The indications for operative correction are to restore or make possible binocular single vision with fusion. Diplopia, head tilting, ocular torticollis, vertigo, nausea, asthenopia, headache, and reflex or remote disturbances, unrelieved otherwise, are indications for operation.

The kind of operation that should be done will depend upon the clinical data in each individual case, and the experience of the surgeon. Theoretically, it is logical to perform a tenotomy on the stronger muscle in cases of excess, an advancement on the weaker in cases of insufficiency, and combined tenotomy for the stronger and advancement for the weaker in mixed conditions. The after effects of a tenotomy are often expressed by a progressive overcorrection, while those following an advancement are seldom subject to that criticism.

The degree of deviation and the rotational power of the eyes should be determined. The latter may be estimated by the position of the vertical

meridian of the cornea when in extreme rotation. The medial rectus should bring the meridian almost opposite the punctum of the lower lid; the lateral rectus should bring it nearly in line with the free border of the lateral commissure of the lids.

The motility is normal in *range* when the excessive excursion in one direction equals the deficient excursion in the other. In such cases an operation is indicated in order to reëstablish the "range" between normal landmarks.

The choice of operation and the eye to be treated are influenced by several factors. In general, tenotomy weakens and advancement strengthens the action of a muscle. The deviating eye should be treated first as a rule. The fellow eye may also require operation. In low degrees of convergent squint due to excess power of the medial rectus, that muscle should be tenotomized.

In higher degrees of alternating convergent squint, convergence may be diminished by an advancement of one or both lateral recti. This operation is also indicated where the lateral recti are weak, or where the vision of the deviating eye exceeds 20/200. The degree of deviation will determine whether the second eye shall be subjected to operation. Usually, where the squint exceeds 15 degrees, both eyes should be treated.

A tenotomy is seldom undertaken where the deviation exceeds 15 degrees, and never where it exceeds 25 degrees, where the apparently overacting muscle does not produce an excessive excursion in its field of action, or where it is unable to perform full rotation even though a squint is present.

A high degree of monocular squint with very low vision in that eye often requires a combination of full correction lenses, tenotomy, and advancement. The glasses should be ordered first. Then a tenotomy is performed on the muscle toward which the cornea is deviated. After the lapse of some months an advancement is performed on the opposing muscle. When these fail to correct, additional muscle operations on the fellow eye are to be considered. It is seldom advisable to combine tenotomy with advancement at one operation, or to operate on both eyes at the same time, except it be to advance both lateral recti at once.

SURGERY OF THE MUSCLES

It is desirable to occasionally test the effects of an operation as it progresses. For that reason local anesthesia is preferable to general. General anesthesia may be used in very young, timid or hypersensitive individuals who cannot be tested satisfactorily before the completion of the operation.

The conjunctival sac is made insensitive with a few drops of 4 per cent solution of cocain and 1 : 5,000 adrenalin, after which a few drops of the same solution or of 2 per cent solution of novocain with adrenalin are injected about

the tendon that is to be reset. When anesthesia is effected, the conjunctival sac is irrigated with 1:5,000 solution of bichlorid of mercury.

Fig. 246.

Tenotomy.—Instruments: Lid speculum (see Fig. 166), toothed forceps, muscle hooks (Fig. 246), muscle scissors (Fig. 247) and needle holder (see Fig. 178).

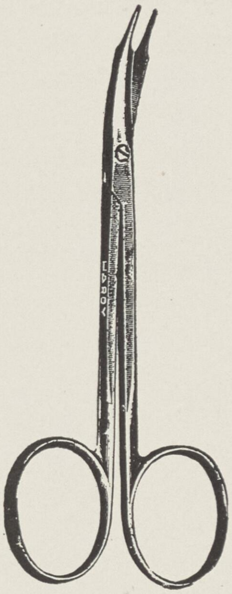


Fig. 247.

The conjunctiva is incised parallel to and 3 or 4 millimeters from the border of the cornea and in front of the insertion of the muscle. The membrane is undermined in the direction of the muscle. The closed forceps are inserted into the pocket until they lie over the tendon; they are then opened, and the tendon is grasped and drawn forward and away from the sclera by elevating the handle of the forceps.

The scissors are held with the convexity of the blades facing the sclera. A nick is made in the lower margin of the tendon at its insertion. This makes an opening in Tenon's capsule. One blade of the scissors is inserted into the opening, and by successive snips the tendon is divided close to the sclera (Fig. 248). The latter must not be injured, and the capsule behind the insertion must not be disturbed. The muscle is released.

A small muscle hook is passed into the wound slightly beyond the site of insertion. It is turned first toward one margin of the muscle and then toward the other. In each position slight traction is made toward the cornea. Uncut tendon fibers resist the pull and must be divided. Capsule fibers impart a tactile sense of elasticity; these must not be cut. When to inspection the eyes appear straight, the patient's glasses are put on him and he is directed to look at distant and near objects. The effects are estimated, and modified if desired. In tenotomy, undercorrection is the rule.

A simplified method of tenotomy is performed as follows: As the forceps are held perpendicularly to the plane of the surface of the eyeball, they are made to pick up the tendon immediately behind its insertion; this causes the tendon and the conjunctiva over it to knuckle up. The center of the knuckle is opened with scissors, close to and in a plane parallel with the surface of the sclera. A muscle hook is inserted into the opening and turned upward. That half of the tendon is slightly lifted and divided subconjunctivally with scissors. The lower half is divided in the same way.

The hook is again inserted and traction is made with it. Rigid fibers are

divided, while elastic fibers are left undisturbed. The conjunctival wound is closed with black silk sutures, which are removed in from five to seven days. White's ointment is applied, and a light dressing is used for two or three days.

Each muscle is supplied by an artery accompanied by two veins. When these are divided, hemorrhage will follow. This does no harm unless the blood flows backward between the sclera and Tenon's capsule. The globe may be forced forward and require a pressure bandage for a few days.

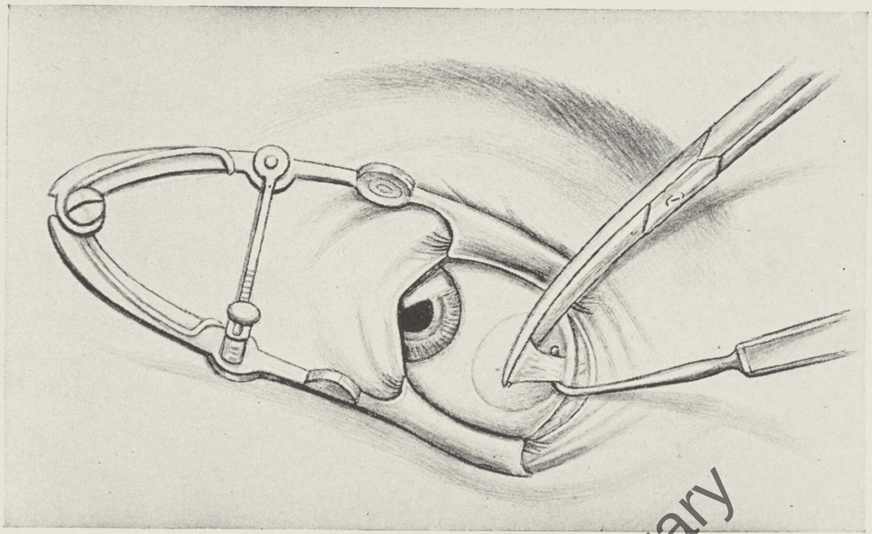


Fig. 248.—Complete Tenotomy.

Fibers from the medial rectus are inserted into the caruncle. This muscle retracts after a tenotomy, and may retract the caruncle with it. This may be apparent at once, or it may be delayed. To restore the caruncle, the conjunctiva should be undermined toward it, for the purpose of dividing the muscle-fibers attached to it. A medicolegal point sometimes rests on this operative complication. An injured individual may claim that in consequence of some accident he has impaired vision in one eye. A retracted caruncle suggests a previous tenotomy for convergent squint, and such a squint is frequently due to amblyopia. The conjunctival scar may have disappeared. The fornices may also be retracted by a free tenotomy.

Undue injury to the fibers of Tenon's capsule is indicated by an overcorrection of the deformity, or by a widening of the palpebral aperture. The muscle may have to be advanced later to modify the overcorrection, and the wide aperture can be remedied by a lateral tarsorrhaphy.

While the immediate result of a tenotomy should be estimated, the final

effect cannot be known for some time. More certain end-results may be had by the employment of sutures to support or to limit the tenotomy.

A *supporting* black silk suture armed with two small curved needles is passed through the conjunctiva and episclera near the temporal end of the horizontal meridian of the cornea, or it may be passed through below and then above the end of the meridian. Both needles are then passed through the tissues of the lateral commissure, and the suture is tied over a gauze roll or metal plate. This is used only when the muscle shows too much strength after tenotomy; it is removed after one or two days.

This suture is also of use within a few days after the operation, when it is seen that the effect of the tenotomy is diminishing. The original wound is opened and the adhesions are broken up with the hook. Then the supporting suture is inserted. While it is a general rule to slightly undercorrect in tenotomies, it is permissible to overcorrect somewhat with the supporting suture.

The *counteracting* or limiting suture is used at once or a few days later, when an overcorrection is noticed. This suture is double-armed. The original wound is opened; the free end of the muscle is found and drawn forward with forceps. Recent adhesions must be broken up. The suture is passed through the end of the muscle from the scleral surface, and then through the superficial tissues adjoining the limbus near the end of the horizontal meridian of the cornea. The ends are drawn until a slight undercorrection of the original position is attained, and are then tied. This suture should remain in place for several days.

Tenotomy of the Inferior Oblique.—An incision is made to the bone along the nasal two-fifths of the inferior margin of the orbit. The muscle is exposed by blunt dissection; surrounding fibrous tissues should be disturbed as little as possible, since fat prolapses readily. Traction is made on the muscle with a muscle hook; this is recognized by movements imparted to the globe. The muscle is divided at its tendon of origin. The wound is closed and a pressure bandage is applied for two days.

This operation is advised in cases of paralytic hypophoria where the superior rectus of the opposite eye is paralyzed, particularly if that eye habitually performs fixation. It is also indicated for spasm of the inferior oblique whenever that condition is responsible for upward deviation or diplopia.

Advancement.—Instruments: Same as for tenotomy.

A vertical conjunctival incision is made as for tenotomy; from this an additional incision is made in the direction of and parallel with the axis of the muscle, and sufficiently long to expose the tendon. The conjunctiva and episclera are undermined over and about the insertion. The tendon is grasped

with forceps and drawn into the field. The muscle is undermined with the closed points of the scissors or the tip of the hook first under one margin and then under the other. A hook is passed beneath the tendon immediately behind the insertion from each side. While one hook is held tightly against the insertion, the other is passed backward until the tendon is free for a distance of 10 to 12 millimeters.

Two silk sutures are armed with thin flat noncutting needles. One needle is passed through the muscle from the scleral surface near the posterior hook, and the suture is drawn partly through. The needle is again passed through near the first insertion so that a loop surrounds that part of the muscle. The

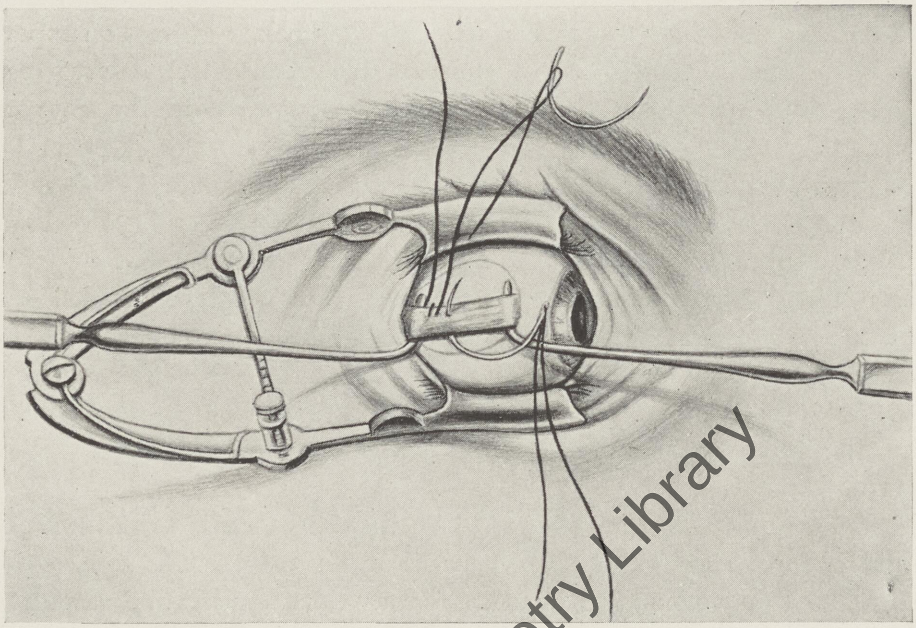


FIG. 249.—ADVANCEMENT. (After Meller.)

other needle is similarly passed twice through the muscle from the opposite side (Fig. 249). The loops are drawn tightly, and the sutures are laid aside above and below, corresponding to the side of the muscle through which they have passed. The muscle is cut off some 2 millimeters anterior to the loops, and the tendon from its insertion into the sclera.

The conjunctiva is undermined toward the limbus. The needle on the upper suture is passed partly through the thickness of the scleral tissue, and then upward by a quilting stitch through the conjunctiva near the limbus (Fig. 250). The remaining needle is passed downward in a similar manner. The tissues here are normally about 1 millimeter thick. In cases of ectasia or blue sclera, where the coat is unusually thin, this operation may be contra-indicated.

The exposed sclera and the under surface of the muscle are freshened to obtain the advantage of raw surfaces for uniting.

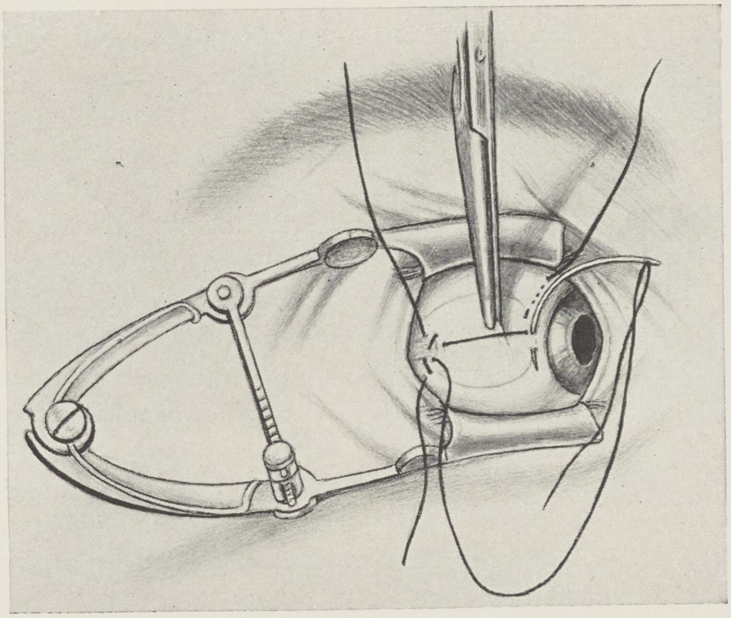


Fig. 250.
(After Meller.)

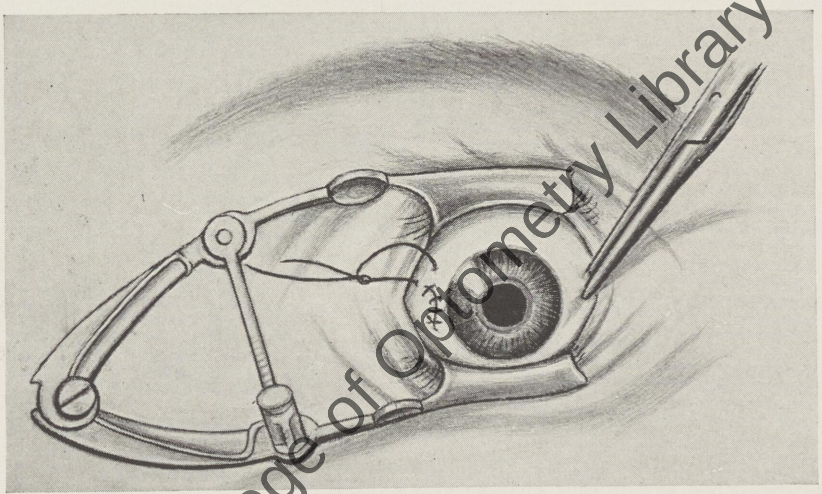


Fig. 251.
(After Meller.)

The cornea is rotated in the direction of the divided muscle (Fig. 251), the sutures are drawn tightly, and the two ends of each suture are tied firmly. The rule is to overcorrect, or "get all you can." An additional suture may

be used to close the conjunctival wound where necessary. White's ointment is applied. A bilateral dressing prevents use of the eyes for four or five days. The sutures need not be removed until all reaction has subsided, unless ulceration of the cornea appears. Where the suture knots are properly located, there is little liability to this complication.

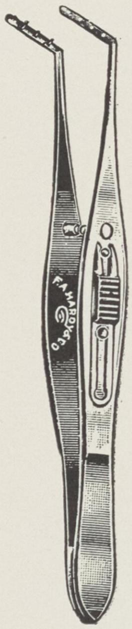


Fig. 252.

Reese's Resection.—Instruments: Speculum, toothed forceps, Hotz's forceps (Fig. 252), muscle scissors, muscle hooks and needle holder.

A vertical incision is made in the conjunctiva and episclera, and those membranes are undermined. A muscle hook is passed beneath the muscle near its insertion, to separate it from its bed. An advancement forceps replaces the hook and grasps the muscle at right angles to its axes 10 or 12 millimeters behind the insertion. The muscle is then divided about 2 millimeters behind the insertion.

A heavy silk suture is double-armed. One needle is passed through the muscle, from the scleral surface behind the forceps, at the junction of the upper and middle thirds, and the other needle at the junction of the middle and lower thirds. Both needles are then passed through the edge of the posterior conjunctival flap. Two fine sutures are passed through the conjunctiva from without inward, and then through the upper and lower borders of the muscle respectively, somewhat posterior to the heavy suture. The muscle is then cut off behind the forceps (Fig. 253).

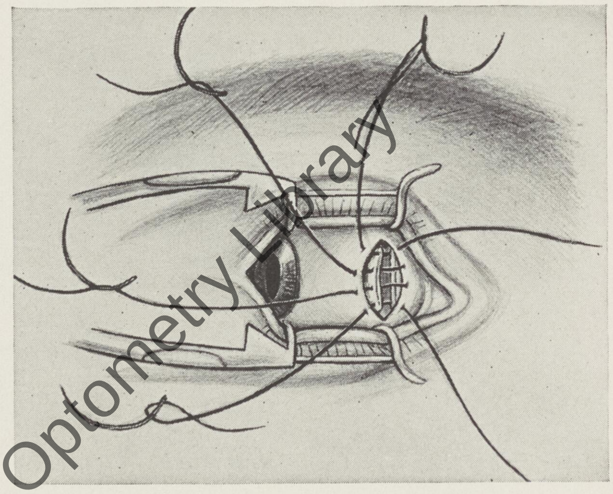


Fig. 253.—Reese's Resection. (After Wheeler.)

The needles on the heavy suture are passed through the stump of the tendon from the scleral side, about 2 millimeters apart, and are brought out through the conjunctiva anteriorly. The fine sutures are passed through the upper and lower borders of the stump from within outward and through the conjunctiva. The heavy suture is drawn tightly and tied. Then the others are tied (Fig. 254). Both eyes are bandaged for three days. The fine sutures are removed

at this time, and only the treated eye is bandaged. The heavy suture is not removed until the tenth day.

Tucking.—Two methods are employed: In one the tuck is made with an instrument designed for the purpose, while in the other the whole correction is made with sutures. The muscle is freed as for advancement, and its conjunctival surface is freshened.

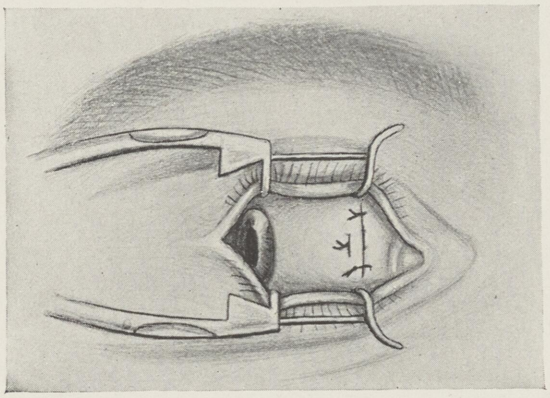


Fig. 254.
(After Wheeler.)

A double-armed suture is passed parallel with the sclera through the tendon at its insertion. One needle is then passed through the tendon from the conjunctival surface half way between its margin and midline; it is next passed through the muscle in a corresponding position from the scleral surface about a centimeter further back, and finally through the conjunctiva from beneath, so that the suture may be tied outside. The other needle is similarly passed twice through the opposite half of the muscle and the conjunctiva.

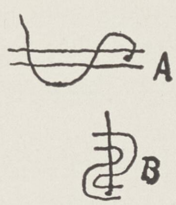


Fig. 255.—Tucking.

When traction is made on the two ends of the suture, the muscle is folded mechanically in the form of a capital S or Z (Fig. 255). The suture is drawn tightly and tied over a metal plate. An additional suture is used to close the conjunctival wound.

Whether made by the special instrument or by folding, a lump of muscle remains beneath the conjunctiva; it gradually flattens.

Advancement may be practiced on any rectus muscle. This operation is applicable to latent as well as to manifest deviations. The operation should not be performed in cases of paralysis, unless the squint is permanent and has been so for many months.

NYSTAGMUS

True nystagmus consists of involuntary conjugate oscillations of the eyeballs. It is seldom monocular. There are two types of movements. In the *undulatory,* vibratory or pendulum type the excursions are equal in velocity and range. In the *rhythmic,* resilient or jerky type the excursions are slow in one direction and rapid in the other; these movements are designated as the slow

and rapid components. According to the rate and range of excursion the nystagmus is also called fine or coarse.

According to the direction of movement, the manifestation is known as lateral or horizontal, vertical, rotary and mixed nystagmus. The oscillations are most frequently horizontal, and less frequently they are vertical or rotary. Mixed forms are combinations of oscillations. Rarely, convergence alternates with divergence, and very rarely the eyes seesaw in vertical movements. Monocular nystagmus is usually vertical.

A latent nystagmus may become apparent when one eye is occluded, or when fixation is attempted. It may not be discovered until the fundus is examined and the disk is observed to oscillate. When nystagmus seems to be monocular, an examination of the fundus of the fellow eye may disclose that the condition is bilateral.

Nystagmus does not interfere with normal rotations and convergence. The oscillations cease during sleep and while the eyes are closed. A physiological or pseudonystagmus usually occurs when the eyes are rotated into extreme lateral positions. Some persons can produce the movements voluntarily. It is often observed among passengers on railroad trains.

Etiology.—Nystagmus occurs secondarily to defects or diseases of the eye, ear, and central nervous system. It is not a primary affection. Several *congenital* conditions produce oscillations. In albinos the nystagmus may be essentially a searching movement made in the endeavor to obtain fixation and to improve vision in bright light. The albino has better vision in dim light; the retinal cones are said to be defective or functionless. In such cases there can be no true macular vision.

In congenital retinitis pigmentosa and in some cases of chorioretinitis, nystagmus may be a searching movement; the vision is poor. Where the maculæ are involved, or where there is macular degeneration, a central scotoma can prevent accurate fixation, and nystagmus may occur. The nystagmus associated with congenital total color-blindness may belong in this group. In acquired macular degeneration nystagmus is usually absent.

Opacities in the media of the eye, such as congenital cataracts and corneal leukomata, interfere with central vision and fixation. A searching nystagmus is often associated. Amblyopia and high refractive errors diminish vision to an extent that no sharp images can be formed on the retina. In the effort to improve vision oscillations may occur. Children who are born blind or who have not developed fixation rarely have nystagmus, unless it is due to disease of the central nervous system.

Spasmus nutans is usually accompanied by nystagmus. The head nodding may be vertical, horizontal, rotary or mixed. In some cases the head has been

observed to move opposite to the direction the eyes move; that is, as the head moves to the right the eyes move to the left. In such instances the nystagmus compensates the movements of the head, and permits a reasonable degree of vision and fixation.

Hereditary influences follow one of two lines: (1) Defects are transmitted to males through unaffected females, as in color-blindness. (2) Other defects, such as albinism, may be transmitted or inherited by either sex.

Acquired Nystagmus may occur as an occupational disease, or it may arise in consequence of some disease of the central nervous system or of the vestibular apparatus. Coal miners are occasional victims. Their susceptibility has been mainly ascribed to the poor illumination in which they work, and their positions while at work. Parsons states that nystagmus is six times more frequent where safety lamps are in use, as compared with pits which use exposed lights. In this form the oscillations are rapid and rotary or irregular; they are most pronounced when looking upward, and may be absent when looking downward. This type seems to be more prevalent in England than in America.

In normal convergence in the reading position the superior oblique muscles assist the medial recti. In convergence upward the medial recti are assisted by the inferior obliques. The latter may become readily fatigued at their unaccustomed and unnatural task, and demand excessive motor impulses in order to meet the strain. While all coal miners do not suffer from nystagmus, it is to be remembered that all individuals are not uniformly susceptible to the injurious effects of eye-strain.

Seamstresses, milliners and typesetters are also subject to the symptom. They often work in poor light and in strained positions. Low illumination taxes fixation. These patients see better in bright light. Fatigue and lowered vitality predispose workers in these occupations.

Nystagmus is observed in some cases of disseminated sclerosis, hereditary cerebellar ataxia, Friedreich's ataxia, paraplegic ataxia, multiple neuritis, syringomyelia, Little's disease, encephalitis, intracranial hemorrhages, emboli, abscesses and tumors, hydrocephalus, idiocy, hysteria, and following some head injuries.

Some poisons may cause nystagmus, but the symptom is without value in identifying the causative agent.

Vestibular Nystagmus may occur in injury or disease of any part of the vestibular apparatus. Fistula of the labyrinth, acoustic nerve tumors, and new growths in the cerebellopontine angle or in the cerebellum are nearly always accompanied by nystagmus.

The behavior of the vestibular system is so constant under fixed conditions that certain tests have been devised and standardized, not only for localizing

intracranial disease, but to determine the reactions of normal individuals. These tests depend upon the movements of endolymph in the semicircular canals. The nystagmus is rhythmic; there is a slow and a rapid component. The effect is diminished when the patient looks in the direction of the slow component, and it is increased when looking in the direction of the rapid component. The rapid component only is abolished by profound anesthesia.

Symptoms.—Vision is poor, especially in the congenital forms; it is worse at night in the occupational forms, but not in albinos. Reading is difficult, and some patients read more satisfactorily with the printed lines held in the vertical position. Photophobia is present in albinism and in miners' nystagmus. Vertigo and dizziness are found in both the miners' and vestibular varieties. An apparent movement of objects annoys those who have diseases of the nervous system or miners' nystagmus, but this is not present in the congenital form. Affected miners often complain of headache, blepharospasm, tremors and mental depression.

Diagnosis.—This is made from the oscillations which are evident to inspection, or which may be discovered with the ophthalmoscope. Nystagmus must not be confused with the aimless movements made by totally blind eyes.

Treatment is directed to the cause. Ametropia should be corrected. Smoked glasses may afford some relief. Muscle imbalance should have appropriate treatment. The eye muscles ought to be rested by closing the eyes occasionally. A cataract should be removed. An optical iridectomy may be performed in a suitable case of corneal leukoma. Eserin locally has benefited some patients. Strychnin internally may be used to near tolerance. Miners should change occupations. Treatment is mostly palliative.

Prognosis.—Spasmus nutans disappears spontaneously, unless it is of the very rare hereditary variety. With a change of occupation, miners' nystagmus is permanently benefited after a time. Little can be expected in cases of disease of the nervous system. When nystagmus is due to permanent conditions such as albinism and chorioretinitis, no cure is possible.

CHAPTER XVII

THE INTRINSIC MUSCLES OF THE EYE

The Iris.—The intrinsic ocular muscles are those of the iris and the ciliary body. The function of the iris is to control the size of the pupil and so regulate the amount of light rays admitted to the retina. The normal pupil varies in size in inverse ratio to the intensity of light, and in direct ratio to the activities of accommodation and convergence.

There are two zones of unstriped muscle-fibers in the iris. A sphincter zone surrounds the pupil; when it contracts the pupil becomes smaller. A zone of radiating fibers extends from the sphincter to the periphery of the iris; when these contract the pupil dilates. The sphincter is controlled by the parasympathetic, and the dilator by the sympathetic. The two systems act in harmony to meet visual requirements.

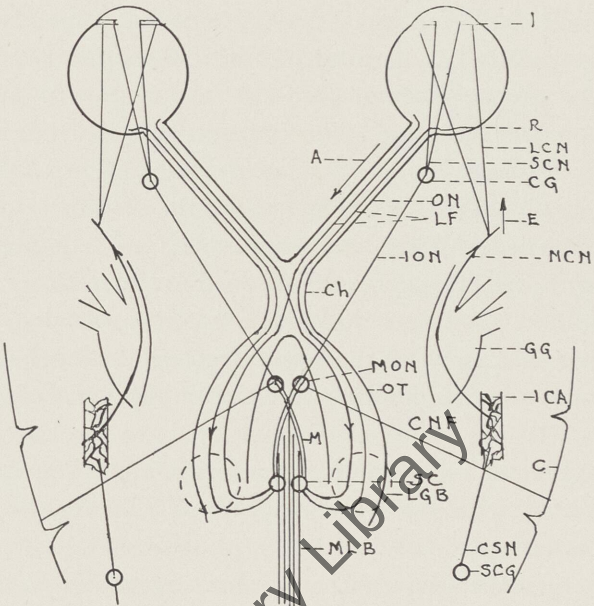


Fig. 256.—*I*, Iris; *R*, Retina; *LCN*, Long Ciliary Nerves; *A*, Afferent Path; *SCN*, Short Ciliary Nerves; *CG*, Ciliary Ganglion; *ON*, Optic Nerve; *LF*, Light Fibers; *E*, Dilating Efferent Path; *ION*, Inferior Oblique Nerve; *NCN*, Nasociliary Nerve; *Ch*, Chiasm; *GG*, Gasserian Ganglion with Three Branches; *MON*, Motor Oculi Nucleus; *OT*, Optic Tract; *ICA*, Internal Carotid Artery with Sympathetic Plexus; *CNF*, Corticonuclear Fasciculus; *M*, Meynert's Fountain Decussation; *C*, Cerebral Cortex; *SC*, Superior Colliculus; *LGB*, Lateral Geniculate Body; *MLB*, Medial Longitudinal Bundle; *CSN*, Cervical Sympathetic Nerve; *SCG*, Superior Cervical Ganglion.

The Sphincter.—From light-sensitive cells in the retina neurons go by way of the optic nerve to the chiasm (Fig. 256). Here the neurons divide into homolateral and contralateral bundles, so that light fibers from both eyes are found in each optic tract. From the tract the fibers pass by the lateral geniculate body and terminate in the superior colliculus. This constitutes the centripetal or afferent path; no lesion posterior to this can affect the light reflex arc.

Fibers arising from cells in the superior colliculus pass to the opposite motor oculi nucleus. Both eyes are represented in each of these nuclei. From cells in the nucleus fibers enter the nerve for the inferior oblique muscle, but leave it as the short root of the ciliary ganglion. From the ganglion the short ciliary nerves are distributed to the sphincter. This constitutes the centrifugal or efferent path. Stimulation of the light reflex arc causes the pupil to contract; inhibition allows it to dilate.

The behavior of the pupils when exposed to light can be understood by attention to the partial decussation in the chiasm, and to that from the colliculi to the nuclei of the motor oculi nerves. When either pupil alone is exposed to light, both contract; the one exposed responds directly, the other indirectly or consensually. A total lesion of the optic nerve on the side exposed interrupts the afferent path on that side and neither pupil contracts. When the other eye is exposed, both pupils contract, provided the nerve on that side is intact.

Should the chiasm be divided anteroposteriorly, or the tract transversely, or one colliculus be destroyed, the response to light will not be abolished, but modified. When any such condition is bilateral, neither pupil contracts. In an optic atrophy that affects the visual fibers only, the light reflex will be retained. A lesion that lies between the nucleus of the motor oculi nerve and the iris will prevent the pupil from contracting on the corresponding side; where such a lesion is bilateral, neither pupil can contract.

In the motor oculi nucleus there are two groups of cells which act on the sphincter; one for light reflex contraction, and one for the contraction associated with convergence. When convergence cannot be stimulated, the associated contraction of the pupil is absent. But the pupils will contract to a convergence impulse, whether convergence is accomplished or not.

Haab's psychic reflex can be demonstrated in a darkened room. The patient looks at a light and then into a darker part of the room, and the pupils dilate. While he continues to look into the dark he thinks of the light and the pupils contract. This reflex has some value in testing the attention in psychiatric patients; in dementia præcox no psychic stimulus affects the pupils.

The Dilator.—The cells of origin for pupillary dilatation are believed to be located in the cerebrum. Neurons pass downward through the brain stem, the medulla and the spinal cord to the ciliospinal center in the region of the lower cervical and the upper thoracic vertebræ. From this center fibers pass through the stellate and inferior cervical ganglia and terminate in the superior cervical ganglion.

New fibers arise and accompany the sympathetic plexus on the internal carotid artery until they are incorporated in the gasserian ganglion; they then go out in the nasociliary nerve, and leave it as the two long ciliary nerves which

terminate in the dilator muscle. Stimulation of the sympathetic dilates the pupil; inhibition allows it to contract, as it does in sleep.

A center for dilatation is probably located in the brain stem, and various stimuli can reach it by way of the spinal cord from the body. The medial longitudinal bundle is an association tract extending from the lower end of the medulla to the region of the nucleus of the motor oculi nerve. It establishes relations between the various cranial nerves. By means of it, dilator influences may reach the nucleus of the motor oculi and inhibit the sphincter when the sympathetic is stimulated. While the parasympathetic is stimulated, the sympathetic is inhibited and the pupil contracts.

The pupillary sphincter is dominated by the cerebrum; cerebral stimulation from the irritations produced by pressure and inflammation is accompanied by miosis. Cerebral inhibition or depression due to toxins permits the pupil to dilate. The dilator is dominated by the spinal center; stimulation at this center produces mydriasis, and paralysis at this level permits the pupil to contract.

When the parasympathetic is inhibited the pupils dilate; when the sympathetic is inhibited the pupils contract. The behavior of the pupil has significance when administering ether or chloroform; the pupils dilate in the stage of excitement, and contract during the period of surgical anesthesia. The gastric emesis reflex indicates that anesthesia is too light; the pupils dilate, but they also dilate when the narcosis is so deep that cerebral impulses are inhibited.

The Clinical Pupil.—In estimating the diagnostic value of an abnormal pupil, diseases of the eye and the effects of drugs must be excluded. In iritis the pupil contracts; in glaucoma it dilates. The pupil is usually dilated in an optic atrophy that affects the light fibers from the retina; it may be contracted from some irritation of the cornea. Mydriatics and miotics are used by malingerers. The pupils should be tested in light that is not too bright, and that is equal for each eye. A magnifying glass or a loupe will aid in the detection of pupillary movements that elude the unaided eye.

When attending head injury cases or other emergency unconscious patients, it is important to note and record the sizes of the pupils. The formerly larger one may be the smaller when the patient regains consciousness. When the pupils are equal in size, their dilatation or contraction and response to light should be recorded. In coma from cerebral pressure the pupils dilate; in uremic coma they contract.

In hemiplegic crossed paralysis the pupil on the side of the unaffected cerebral hemisphere will react normally. During coma it will contract as it does in sleep, but it will dilate when the patient is conscious. By reason of the paralysis on the side of the affected hemisphere, that pupil will not react

normally. It reacts sluggishly or it may be immobile. When compared to the pupil on the unaffected side it will *appear* to be dilated during coma and contracted during consciousness.

In the early stages of acute bilateral meningitis the cerebrum is irritated and the pupils contract; in the developed stage the pupils dilate from cerebral paresis. This is especially true in serous meningitis of the base, in the region where the nucleus and fasciculus of the motor oculi nerve are most exposed to inflammatory products.

Immobility.—In *reflex* immobility the pupil reacts to convergence but not to light. In *absolute* immobility it reacts to neither; paralysis of accommodation may or may not be associated. Syphilis is prone to attack the tissues around the cerebral aqueduct and the third ventricle. Lesions associated with the Argyll Robertson pupil have been found in this region. When they involve the fibers from one colliculus to the opposite motor oculi nucleus, one light reflex arc is broken; where they involve both paths, both arcs are broken. Lesions above these nuclei do not inhibit contractions to convergence. When the nuclei are involved the pupils cannot contract to light or convergence.

Spinal miosis is due to a destructive lesion in the ciliospinal center. The pupil is small; it reacts to convergence but not to light. It is in a state of reflex immobility. Although one pupil may be affected before the other, it is practically always bilateral. This is the earliest characteristic sign in tabes.

This type of pupil may precede the stage of absolute immobility in general paralysis, especially when there is inequality in the size and reactions of the pupils. In this disease the pupils may become widely dilated as well as absolutely immobile. They will eventually fail to react to convergence. The convergence reflex is preserved much longer in tabes.

In syphilis of the nervous system absolute immobility of one pupil is the rule, and paralysis of accommodation will probably be associated with it. In the last three diseases pupillary signs are valuable; they are not reliable in diffuse diseases of the brain and cord.

Where the Argyll Robertson pupil is associated with a disease other than tabes, the lesion is probably in the region mentioned whether syphilitic or not. In tabes the miosis comes on early and is often followed by primary atrophy of the optic nerve, which accounts for the acquired blindness. In nontabetic atrophy the pupils will likely be dilated and immobile. In hysterical amblyopia with dilated immobile pupils, optic nerve atrophy is absent and the cornea has diminished sensibility.

Congenital miosis and senile miosis should not be mistaken for something serious. A few cases have been recorded in which there were defective convergence and loss of the pupillary convergence reaction, but preservation of the

light reflex. They have been mentioned in connection with various lesions of the central nervous system. It was observed in a case of tobacco amblyopia.

In general, poisons irritate the brain early and paralyze it late. In chronic alcoholism the pupils are small and react sluggishly. In the acute form the pupils will be governed by the excitation or depression present, and whether the cerebrum or cord is affected. Morphin and opium addicts have small but reacting pupils. In axial neuritis the pupils dilate as vision fails. The pupils are immobile in the forms due to quinin and antipyrin, but they react in the form due to tobacco, as they do in acute retrobulbar neuritis.

The pupil dilated by a mydriatic is distinguished from that dilated from amblyopia when the patient can read print through a pinhole in a card. Following diphtheria there may be a paralysis of accommodation with or without immobile pupils. Paralysis of accommodation has been observed in ptomain and lead poisoning, and after influenza. Bilateral widely dilated immobile pupils of recent origin, with paralysis of accommodation, dysphagia, and a history of questionable food suggest botulism. The same ocular phenomena are sometimes found in lethargic encephalitis.

The earliest stage of a degeneration may be irritating. Where this occurs at the level of the ciliospinal center it will stimulate the dilator fibers. As destruction of tissue progresses, by erosion or sclerosis, the dilator impulse cannot be transmitted and the pupil contracts. Subtentorial and pontile tumors irritate the dilating fibers in the medulla, and the pupil is dilated corresponding to the side irritated. Hemorrhage into the pons paralyzes this tract and the pupil contracts. Where but one pupil is affected, it may be due to stimulating or inhibiting pressure on the sympathetic by aneurysm of the aorta or other vessel, tumor of the thorax or neck, cervical rib, neck wound and so forth.

A swelling of the bronchial glands, with the pupil dilated on the same side, may be the earliest sign of pulmonary tuberculosis, in children especially. Intracranially there are encephalitis, tumor, hemorrhage, softening, abscess, cyst, or degeneration to account for pupillary phenomena. Intestinal worms or other irritations may cause the pupils to dilate.

The pupils often react sluggishly when vision is diminished. They may dilate from physical exhaustion, or contract spasmodically after prolonged near work. A foreign body on the cornea can cause miosis.

The Ciliary Body.—Three sections of muscle-fibers have been identified in the ciliary body (see Fig. 288). A meridional or longitudinal section lies next the sclera; it extends from the region of the pectinate ligament anteriorly to the choroid posteriorly. This part is called Brucke's or Bowman's muscle (*B*). A section of circular fibers surrounds the equator of the lens; it is called

Müller's muscle (*M*). This should not be confused with the involuntary orbital or lid muscles which also bear the name of Müller. The third section is constituted of radially disposed fibers.

The ciliary muscle as a whole may have something to do with aqueous drainage. The principal function has to do with accommodation. As the circular muscles contract, the zonular fibers are loosened and the lens becomes more spherical from its own elasticity (Helmholtz's theory). When the function of accommodation is suspended by a cycloplegic, the circular fibers are inhibited. Impulses for accommodation are carried by the motor oculi nerve through the ciliary ganglion; pupillary contraction and convergence are associated with accommodation.

Nothing definite is known of the function or innervation of Brucke's muscle. Savage states that it is innervated by the sympathetic nerve, and that its function is to compensate corneal astigmatism; this it is able to do when certain bundles only of its fibers contract.

The behavior of the ciliary muscle has little clinical or diagnostic importance in general disease. The incidence of paralysis of accommodation has been mentioned in connection with paralysis of the pupil.

CHAPTER XVIII

REFRACTION

THE ESSENTIALS OF OPTICS

A luminous body emits light rays which travel in straight lines in every direction at the same velocity as long as they remain in media of the same density. While all light rays diverge from the source, those which enter the eye from a distance of 6 meters (20 feet) or more are assumed to be parallel.

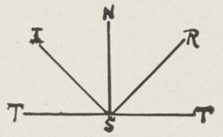


Fig. 257.—*TT*, Surface.

The velocity of light is accelerated or retarded by passing from a medium of one density into that of another according to the relative densities of the two media. When rays of light meet an opaque substance they are absorbed or reflected. When they meet the surface of a transparent medium, part of the rays are reflected and the remainder are refracted by the new medium.

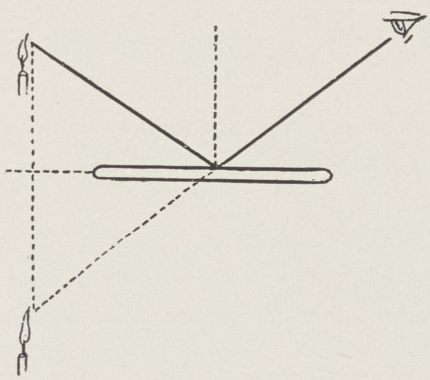


Fig. 258.

The light ray which meets a surface is an incident ray; that part of it which is reflected is a reflected ray, and the part that is refracted is called the emergent ray as soon as it has passed through the interposed medium. A normal is a line drawn perpendicularly to a surface; a ray propagated along the path of a normal is not reflected or refracted.

The incident (*IS*) and reflected (*SR*) rays are in the same plane with the normal (*NS*), and the angles that each makes with the normal are equal (Fig. 257). The critical angle is an exception.

MIRRORS AND IMAGES

Rays from a luminous source are reflected from a plane mirror (Fig. 258). An observing eye situated in the path of the reflected ray will experience the same sensations it would have in looking at the source of light itself. In any

other position the eye will see the image of the luminous object as far behind the mirror as that object is actually in front of it.

For a concave mirror, the normal is represented by a radius, or a line connecting the center of curvature with the circumference. This is the principal axis. When parallel rays of light, which are also parallel with the axis, are reflected from the mirror, they meet on the axis at the principal focus.

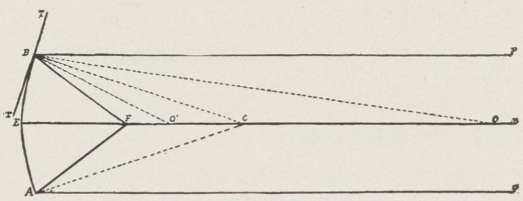


Fig. 259.

In Figure 259, *AEB* represents a concave mirror; *C* is the center of curvature; *CB* is a radius which is normal to the tangent *TBT*; and *DE* is the principal axis. The principal focus is at *F*. The distance from *F* to *E* is the focal length of the mirror. Parallel rays of light are represented by the lines *PB* and *P′A*. The angles *PBC* and *CBF* are equal, as are the angles *P′AC* and *CAF*. Then the reflected rays are brought to a focus or intercept each other at *F*.

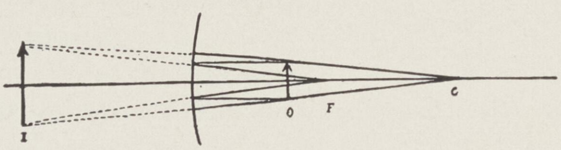


Fig. 260.—*O*, Object; *I*, Image; *F*, Principal Focus; *C*, Center of Curvature.

The rays from a luminous point *O* will be focused at *O′* after reflection. And rays from *O′* will focus at *O*. The angles *OBC* and *CBO′*, and the angles *OAC* and *CAO′* are equal. The points *O* and *O′* are conjugate foci, since they bear a reciprocal relation to each other. The rays from a luminous point situated at *F* will be reflected along the lines *BP* and *AP′* as parallel rays. Parallel rays can form no focus.

Three kinds of images can be formed by a concave mirror. In Figure 260, a dart is situated between the mirror and its principal focus. Lines which are parallel to the principal axis are drawn from either extremity of the dart to the mirror. Lines from the center of curvature are drawn through either extremity of the dart to the mirror. The parallel lines are reflected back to the principal focus. The lines from the center of curvature and from the principal focus are projected behind the mirror. Where these lines intersect, an image of one end of the dart will be formed; lines from all points of the dart are similarly projected. The image will be erect, virtual and enlarged.

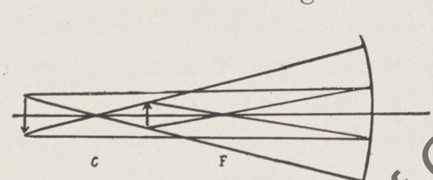


Fig. 261.

In Figure 261, the object lies between the center of curvature and the principal focus. Lines drawn from either extremity of the dart through the princi-

pal focus are reflected from the mirror parallel. Other lines pass from the center of curvature through either end of the dart; where these and the parallel lines intersect, an enlarged inverted image is formed.

In the same figure the object may lie beyond the center of curvature. Parallel lines drawn from either extremity of the dart are reflected through the principal focus, and where these intersect the lines drawn through the center of curvature, a diminished inverted image is formed. In this figure the image and the object are conjugate.

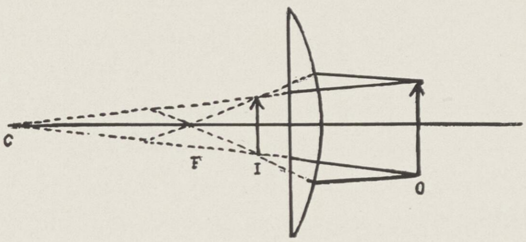


Fig. 262.

Images formed by a convex mirror are always virtual, erect and diminished. In Figure 262, the intersections of the broken lines denote the position of the image (*I*).

PRISMS

A ray of light passing obliquely from a rarer to a denser medium is deflected toward the perpendicular of the surface of the denser; when it passes from a denser to a rarer medium, it is reflected from the perpendicular. The normal ray is not deflected. In Figure 263, the perpendicular ray *AB* is straight. The ray *CD* is partly reflected toward *E*, and partly refracted *toward* the perpendicular *FG* in passing through the denser medium. Upon emerging, *CD* is again refracted, but this time *from* the perpendicular. After the second refraction, *CD* is propagated parallel to its original course or toward *H*.

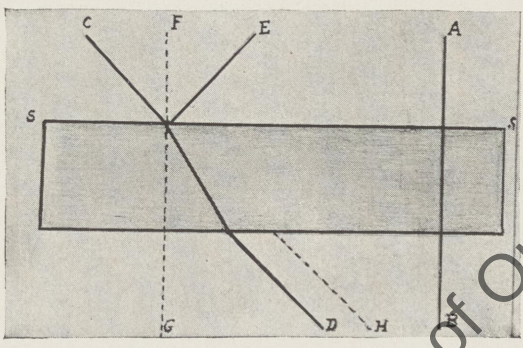


Fig. 263.—*SS*, Surface of Medium.

The deflection is not the same for all media, since density varies for different substances. A vacuum is the standard, but air is so little higher that it is ordinarily considered as 1. Water is 1.33, crown glass is 1.51, flint glass is 1.58, and so on. This relative density is called the "index of refraction."

Where both surfaces of the denser medium are parallel planes, the emergent ray will be propagated parallel to its original course. Where these surfaces are not parallel, they are necessarily convergent in one direction and divergent in the other. This is the essential feature of a prism.

An optical prism consists of a wedge-shaped piece of glass having two plane surfaces which meet at one edge, called the apex, and which terminate opposite the apex in a thick rectangular portion called the base (Fig. 264). The power of a prism depends upon the angle of divergence of its surfaces, and the refractive index of the substance from which it is made.

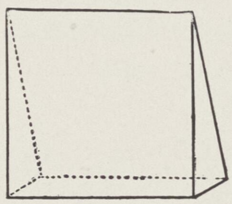


FIG. 264.

Incident light rays meet the first surface of a prism and are deflected toward the perpendicular of that surface, unless they enter perpendicular to it; emergent rays are deflected from the perpendicular of the second surface, unless they emerge as perpendiculars. Both incident and emergent rays are deflected toward the base of a prism, unless one of them is perpendicular to the surface on its own side (Fig. 265). Parallel incident rays pass through a prism and emerge as parallel rays (Fig. 266).

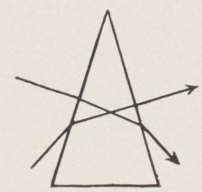


FIG. 265.

The effect of a prism is to apparently displace an object toward the apex; in Figure 267 the object o appears to be situated at o'.

The deflecting power of prisms is indicated in degrees, dioptries or diopters, and centrads. The first method indicates the number of degrees of arc that a prism will deflect a light ray. The dioptric method employs a tangent erected perpendicularly from the radius of a circle (see Fig. 268, A). Where the radius and tangent are each 1 meter (40 inches) long, the tangent line is marked in centimeters. Each unit of prism will deflect the ray 1 centimeter or 1 per cent of the length of the tangent line. The symbol for the prism diopter is the Greek letter delta.

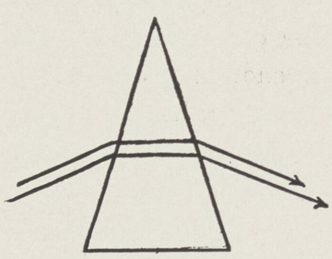


FIG. 266.

The symbol for the centrad is the inverted Greek delta. A radian, which is an arc of a circle equal in length to the radius of that circle (see Fig. 268, C), is marked off into one hundred parts. In this method the unit of deflection is 1/100 or 1 per cent of the radian. For the thin prisms used in ophthalmic practice the last two methods are used interchangeably up to 20 diopters or centrads.

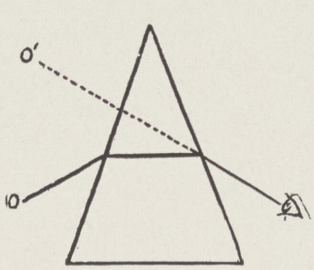


FIG. 267.

The strength of a prism may be ascertained in one of two ways. When two prisms of equal power are superimposed base to apex and apex to base they are neutralized. By superimposing trial case prisms

of different strengths in reversed position, the one which neutralizes the unknown equals it in power.

The axis of a prism is a line equally distant from and parallel with the apex and base. In looking through a prism at a distant line in such a manner that the axis of the prism is parallel to the line, and only a part of the line is seen through the glass, that part appears to be displaced toward the apex of the prism; the line appears to be broken at each edge of the glass (see Fig. 275). When the prism is neutralized, the line will be continuous.

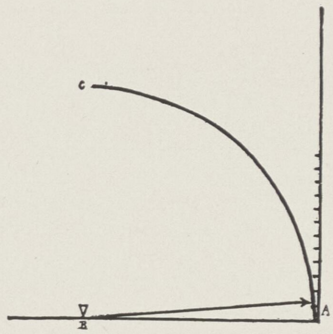


Fig. 268.

The actual displacement effected by a prism may be ascertained. One of several scales may be used; that originated by Ziegler is good (Fig. 269). The prism is held apex up or to the left before the lower right corner of the scale. The power is indicated by the number of intervals of displacement. For testing at a distance of 1 meter (40 inches) the intervals between the lines should be 1 centimeter wide; each interval will represent one unit.

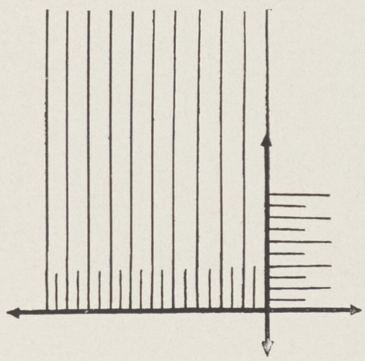


Fig. 269.—Prism Scale.

Prisms are used (1) to determine the amount of latent or manifest deviation of the visual axes in phorias or squints, (2) to ascertain the power of extra-ocular muscles, (3) to exercise weak muscles, (4) to compensate diplopia and restore binocular single vision, and (5) to assist in the detection of malingerers. These uses are discussed in the section on the extrinsic muscles.

CYLINDERS

Two rectangular prisms of equal power are cemented together base to base (Fig. 270). Let each of the two sides be ground into a single curved convex surface (Fig. 271) like the curved surface of a section cut from a solid cylinder (see Fig. 272, *A*). This forms a biconvex, plus or positive cylindrical lens.

A ray of light which passes through the base plane of the double prism, or through a corresponding part of the cylinder is not deflected or refracted, because it passes through both bases and cannot be deflected toward either; it is in the position of the principal axis (see Fig. 259). The *axis of a cylinder* is a line equally distant from, in the same plane, and parallel with both edges

of the cylinder. The *principal axis* is any line which passes perpendicularly through both the axis and the plane in which it is located. Incident rays which

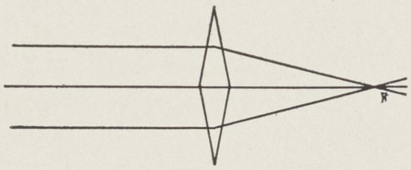


Fig. 270.

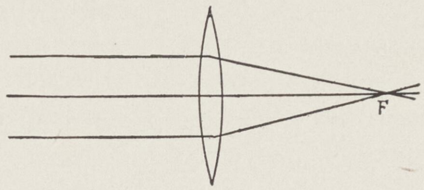


Fig. 271.

pass through the glass in the meridian of the axis are not refracted, while all others are.

Incident rays that are parallel with a principal axis (see Fig. 271) and in

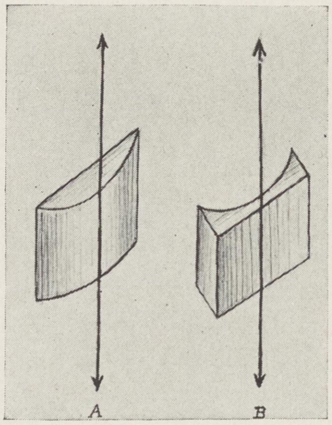


Fig. 272.

the transverse meridian are refracted toward the base of the double prism or toward the principal axis of the cylinder; the emergent rays converge and intercept each other at the principal focus. Incident rays from a luminous point

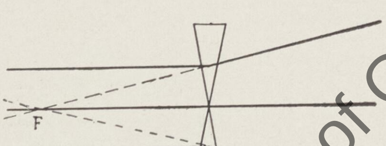


Fig. 273.

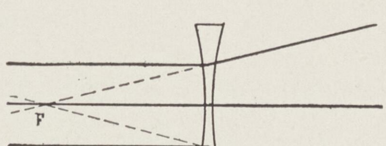


Fig. 274.

situated at the principal focus will be refracted by the double prism or by the cylinder and will emerge as parallel rays.

Two rectangular prisms are cemented together apex to apex (Fig. 273). Let the surface on each side be ground into a single concave curved sur-

face (Fig. 274) like the curved surface of a section cut from a hollow cylinder (see Fig. 272, *B*). The glass will be a biconcave, minus or negative cylinder. The ray which passes through the combined apex of the double prism, or through a corresponding part of the cylinder is not refracted.

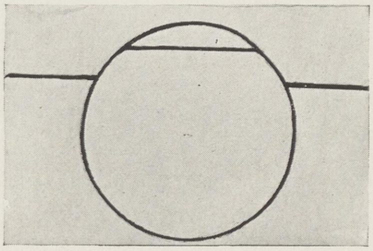


Fig. 275.

Incident rays parallel with the principal axis (see Fig. 274) and in the transverse meridian are refracted away from the principal axis. They emerge divergent, but when projected backward they intercept each other at the principal focus on the opposite side of the lens; this is a negative focus.

A cylinder refracts all rays except those passing through it in the meridian of its axis. Refracted rays are focused into a *line* by a convex cylinder, and are made divergent by a concave cylinder. The negative focus will be represented by a line also.

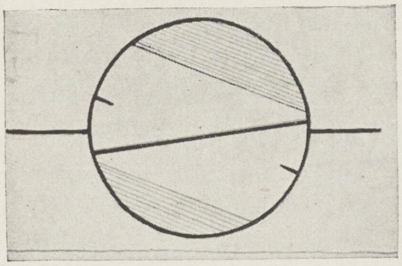


Fig. 276.

Cylinders of opposite value neutralize each other when of the same relative power, plus and minus. The neutralization is effected by superimposing one on the other, axes parallel. An object is always apparently displaced toward the thinnest part of a cylinder, which corresponds to the apex of a prism.

A cylinder is held between the eye and a distant line so that the axis of the cylinder and the line are parallel, and only a part of the line can be seen through the glass. When looking through the upper half of a convex, or the lower half of a concave cylinder, that part of the line seen through the glass will be displaced upward (Fig. 275). By moving the cylinder vertically, the line apparently moves in the opposite direction to the movement of a convex cylinder, and in the same direction if the cylinder is concave.

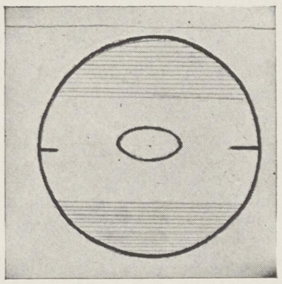


Fig. 277.

When the cylinder is held at an angle to the line, the latter will have the appearance as shown in Figure 276. The unknown axis of a cylinder can be ascertained by rotating the cylinder until the line is unbroken. When looking at a round object like the letter O through a cylinder, but one diameter of the object is diminished by a concave cylinder (Fig. 277) or enlarged by a convex cylinder. A cylinder refracts only at right angles to the meridian of its axis.

The power of a cylinder is designated by diopters, and the unit is that power which will refract parallel rays of light to a focal *line* at a distance of 1 meter (40 inches) from the cylinder. The power of a cylinder is ascertained by neutralizing it with opposite cylinders from the trial case. The displacement of the line, as previously mentioned, may be used for the demonstration.

In the trial case, cylinders are marked with a scratch at each end of the axis or with frosting (see Fig. 276) on each side parallel with the axis. Cylindrical lenses are used for the correction of astigmatism.

SPHERES

An infinite number of triangular prisms of equal power are cemented together, so that all bases are located at a common point called a center; the apices will describe a circumference. Let the surface on each side be ground into a section of a solid sphere. This constitutes a biconvex, plus or positive spherical lens.

A light ray passing through the center of the lens perpendicular to its surface is not refracted, since it is in the position of the principal axis. All other incident rays are refracted. Parallel incident rays that are also parallel to the principal axis in any meridian are refracted toward the principal axis of the lens, and they intercept each other at the principal focus. The distance between the lens and the principal focus is the focal length of the lens.

Incident rays from a luminous point situated at the principal focus will be refracted by the sphere and emerge as parallel rays. Incident rays from a luminous point situated between the lens and its principal focus will emerge divergent. When they originate from a point beyond the principal focus they will emerge convergent, and eventually intercept each other on the principal axis. When they originate at the latter position they will be refracted, and converge beyond the principal focus. These points are conjugate foci, since they bear a reciprocal relation to each other. A lens has an anterior and a posterior principal focus.

An infinite number of triangular prisms of equal power are cemented together, with all apices located at a common point called a center. The bases will describe a circumference. Let the surface on each side be ground into a section of a hollow sphere. This constitutes a biconcave, minus or negative spherical lens.

A light ray passing along the principal axis is not refracted. Parallel incident rays that are parallel to the principal axis are refracted away from that

axis, and hence are divergent. When these rays are projected backward they will converge on the principal axis on the side of incidence, and constitute a negative focus.

An oblique incident ray is refracted toward the principal axis of a convex lens; the emergent ray is refracted away from that axis, but is parallel with the projected incident ray. The ray has been displaced (Fig. 278). In traversing the lens, the ray passes through the *optical center*. A light ray that passes directly through the optical center is not appreciably refracted.

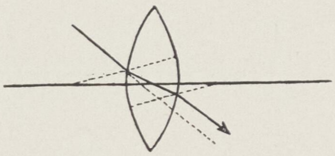


Fig. 278.

The formation of an image by a biconvex spherical lens is illustrated in Figure 279. The ray PE is parallel with the axis Bb; it is refracted through the posterior principal focus O_2 toward P'. The ray PC passes through the anterior principal focus O_1, and is refracted parallel with the axis Bb toward P'. The ray PP' passes through the nodal point N and does not undergo refraction. The image P' of the object P is formed where any two of these three rays intersect. An image of any other point of the object is formed in a similar manner.

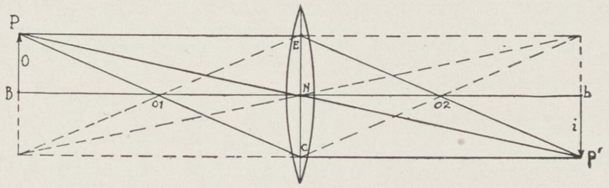


Fig. 279.

These images are real and inverted. When the object is situated at about twice the distance of the principal focus of the lens, the image is the same size as the object; when the object approaches the lens, the image is enlarged; when it recedes, the image is diminished. Considering the lens as stationary, the object and the image move in the same direction, but not at the same speed; they represent conjugate foci. An enlarged erect image is formed by a convex sphere when it is used as a magnifying glass.

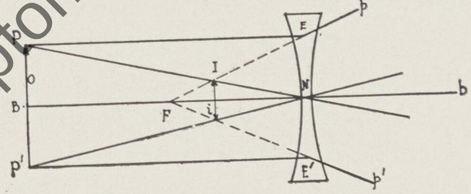


Fig. 280.

Indirect ophthalmoscopy depends upon real, inverted and enlarged images of the fundus oculi.

The formation of an image by a concave spherical lens is illustrated in Figure 280. The rays PE and $P'E'$ are parallel with the axis Bb. They are refracted divergently from the axis toward p and p'. The rays PN and $P'N$ pass through the nodal point and are not refracted. The rays pE and $p'E'$ are projected backward to F. Where these rays intercept the rays PN and $P'N$, an erect, virtual and diminished

image (Ii) of the object PP' is formed. This image can be seen only when looking through the lens at the object.

The power of a spherical lens is indicated by its ability to refract parallel rays of light. The unit of measurement is a lens whose principal focus is 100 centimeters (40 inches) distant; it has a power of one diopter, the symbol for which is D. The focal length of a lens is calculated by dividing the distance in centimeters or inches by the known power of the lens; the power is calculated by dividing the distance by the known focal length. A 5 D. lens has a focal length of 20 centimeters (8 inches).

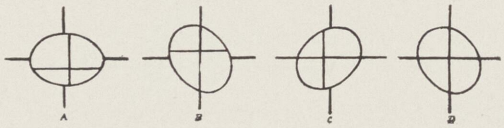


Fig. 281.

The value of an unknown sphere is ascertained by moving it between the eye and a distant object. When the object appears to move in the same direction the lens is moving the sphere is concave; when the object appears to move in the opposite direction the sphere is convex.

Spherical lenses are used to correct ametropia in which all meridians of the dioptric system of the eye are equal; these errors are simple hyperopia or simple myopia. Cylinders are added to spheres when the meridians are unequal, since the inequality constitutes astigmatism.

The optical center of a lens may be located by looking through the lens at lines crossed at a right angle (Fig. 281). The lines are displaced as at *A*; the lens is moved until the vertical line or the horizontal line is continuous as at *B* or *C*; the lens is moved again until both lines are continuous, as at *D*. The optical center is located at the intersection of the lines as shown at *D*.

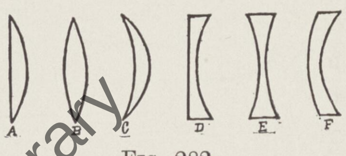


Fig. 282.

The optical center does not always coincide with the geometrical center of a mounted spectacle lens. Lenses are deliberately "decentered" to bring the optical center in line with the visual axis, or to obtain the effect of a prism. (See page 371.)

Ophthalmic lenses are ground in various forms (Fig. 282). In order from *A* to *F*, the cross sections illustrate planoconvex, biconvex, concavoconvex, planoconcave, biconcave and convexoconcave forms. Toric lenses are represented at *C* and *F*.

VISUAL OPTICS

The eye comprises the optical part of the visual system. The function of the optical system is to project the image of an object on the retina. The image is transmitted to the visual centers for interpretation. A confused interpre-

tation may be due to a blurred retinal image; that image is blurred because rays of light from the object are not sharply focused on the retina.

The optical properties of the eye are acquired from its refracting surfaces and media, and from its length. The surfaces are the anterior surface of the cornea and both surfaces of the crystalline lens; the media are the cornea, aqueous humor, lens and vitreous humor.

The media and curved surfaces of the eye are assembled to form a compound dioptric or lens system. The principal axis of this system corresponds to the optic axis, and very nearly to the line of vision; the latter extends from the fovea through the nodal point of the system to the object. Light rays from all points of an object must pass through the nodal point or optical center to form an image of the whole object. The nodal point is the position through which all convergent rays pass, and the angle of arc subtended by the object is the visual angle for that object.

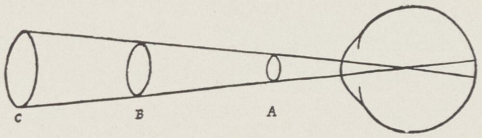


Fig. 283.

The size of the object and its distance from the nodal point determine the size of the retinal image. A small object near the eye may have the same visual angle as a large object situated at a distance, and therefore the retinal images are equal (Fig. 283). The smallest angle in which an object can be recognized by the average normal eye is the minimum or limiting angle of vision; this is an angle of arc of about 1 minute.

The acuity of vision is ascertained by means of standard test letters or characters arranged on cards for the purpose (see Fig. 292). These letters are constructed from mathematical formulæ for the distances they represent. Standard acuity is the ability of the average normal or emmetropic eye to recognize a letter which subtends an angle of 5 minutes in both height and width. The letters are constructed on a square block of twenty-five component squares (Fig. 284). Each square measures 1 minute in each plane dimension. At 6 meters (20 feet) the standard letter occupies a block which is 8.724 millimeters square.

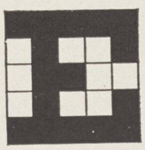


Fig. 284.

The size of the retinal image is to the size of the object as the distance of the image is to the distance of the object in millimeters from the nodal point. The distance of the image from the nodal point is about 15.61 millimeters.

Light rays from a small object situated at a distance of 6 meters (20 feet) or more are approximately parallel. The average normal eye obtains an image of an object at this distance without effort, because parallel incident rays pass-

ing through the dioptric system of the eye at rest are focused on the retina. When the object is situated at a distance less than 6 meters (20 feet) rays from the object are divergent and will be focused behind the retina without effort. Conjugate foci move in the same direction; the nearer the object is to the eye, the farther will its image appear behind the retina.

Accommodation.—The mechanism by which the emmetropic eye compensates for divergent incident rays or those originating from a point nearer than infinity is known as accommodation. The essential feature of accommodation is the lengthening of the anteroposterior diameter of the lens (Figs. 285 and 286). By accommodation the lens increases its refractive power to an extent that the position of the posterior principal focus of the eye is conjugate to the position of the object.

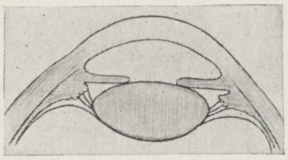


Fig. 285.—Lens at Rest.

Accommodation is effected by the action of the ciliary muscle. According to the theory of accommodation given by Helmholtz the ciliary muscle contracts and relaxes the zonular fibers; the lens thickens anteroposteriorly because of its own elasticity. The motor impulses to the ciliary muscle regulate the amount of accommodation. By a concerted action of the iris, the pupil contracts and excludes the aberrant rays by limiting the number of peripheral rays which enter the eye.

The power of accommodation is called the amplitude of accommodation. This indicates the difference between the dioptric power of the eye while in a state of rest and its power while in maximum accommodation. In a state of rest the eye is adapted for its far-point, which in the emmetropic eye is at infinity or an immeasurable distance. At maximum accommodation, the nearest distance at which the vision is clear is called the near-point or punctum proximum, for which the symbol is P.p.

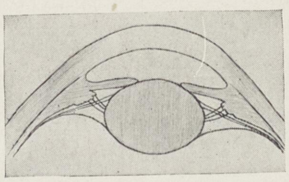


Fig. 286.—Lens in Accommodation.

Presbyopia.—The elasticity of the lens gradually decreases from childhood to old age; as age advances the near-point recedes. The amplitude in diopters is found by dividing 100 by the distance of the near-point in centimeters. The table on page 355, compiled from the calculations of others, is representative for five-year periods; the figures apply to emmetropic eyes, and those made emmetropic by appropriate lenses.

The near-point of accommodation can be tested as follows: One eye is occluded; a Prince's rule is held against the side of the nose with one end about 11.5 millimeters in front of the cornea, or it may be held against the cell of a trial frame. A card of fine well-lighted type is held close to the eye,

and is then gradually drawn away along the edge of the rule. When the patient is able to read the type, the distance is read off from the graduations on the rule; this is the near-point. The other eye is tested in the same way.

Age	P.p., centimeters	Age	P.p., centimeters	Age	P.p., centimeters	Age	P.p., centimeters
10.....	7.0	30.....	12.0	50.....	45.0	70.....	400.0
15.....	8.0	35.....	14.0	55.....	55.0	75.....	infinity
20.....	9.0	40.....	18.0	60.....	100.0		
25.....	10.5	45.....	26.0	65.....	150.0		

By the table it is noticed that during the fifth decade of life the near-point recedes beyond 33 centimeters, or the average reading distance. This marks the beginning age of presbyopia, and the emmetropic patient will thereafter require glasses for near work.

Ametropia.—The eye at rest is emmetropic when parallel rays of light from an object are focused on the fovea; that is, when the image of an object is sharply defined at the fovea. Ametropia is caused by an optical disarrangement that prevents the image from being distinct at that position. Ametropia is considered as axial, curvature, and index, or a combination of these.

Fig. 287.

In *axial* ametropia the fovea lies nearer to or farther from the nodal point than the posterior principal focus; the eyeball is either too short or too long, and the image at the fovea is diffuse or blurred. In *index* ametropia, the index of refraction of the media as a whole is above or below that of normal media; in that case the posterior principal focus falls in front of or behind the fovea, and again the image is diffuse or blurred.

Curvature ametropia is brought about by a lengthening or shortening of the radii of curvature of the several refracting surfaces of the eye. When these radii are lengthened the surfaces are less sharply curved, and there is a loss in refractive power; when the radii are shortened, the curvature is greater, and there is a gain in refractive power. The posterior principal focus lies behind the fovea when the radii are too long, and in front of it when the radii are too short.

Eyeballs that are too long or too short are common (Fig. 287). Index ametropia is rare; it generally depends upon a lens whose nutrition is disturbed as occasionally happens in diabetes. An increase in blood sugar is often attended by a transient myopia, and a rapid reduction of blood sugar is frequently accompanied by a rapid temporary increase in hyperopia.

Curvature ametropia is common. Usually the curvatures of the several surfaces vary in different meridians in the same eye. In refraction, there are two meridians which are at right angles to each other; they are called the first and second principal meridians. The radius of curvature of the vertical corneal meridian may be longer or shorter than that of the horizontal meridian. The same statement may be made in regard to the surfaces of the lens. Such differences in curvature are generally exhibited as astigmatism. An exaggerated idea of such curves is exhibited in trick mirrors.

Hyperopia is an optical state in which the retina lies anterior to the posterior principal focus of the dioptric system of the eye (see Fig. 287, *H*); the hyperopic eye is sometimes called a "short eye." Since the posterior focus is a conjugate focus, rays originating from that point will be projected parallel into space. When that focus is at the fovea, visual rays are projected parallel; but when that focus is behind the fovea, rays from the latter will be projected divergently and can never intersect (see Fig. 298).

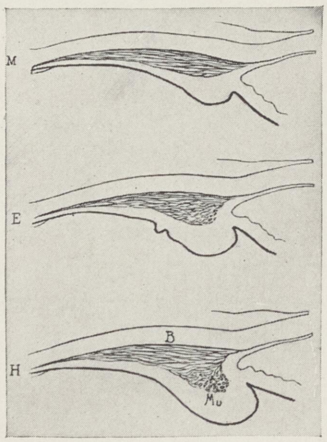


Fig. 288.—*B*, Bowman's Fibers; *Mu*, Müller's Fibers in Cross Section. (After Fuchs.)

The hyperopic eye is required to accommodate for all distances, so that the focal point for parallel rays will be advanced to the retina (see Fig. 287, *E*); in such eyes the ciliary muscle is well developed (Fig. 288), *H*, as compared to the muscle of the emmetropic eye, *E*, or that of the myopic eye, *M*. In the drawing there should be no great difference in the longitudinal fibers, but those shown in cross section at the tip of the process are heavy in *H*, present in *E*, and apparently absent in *M*. Regardless of the strength of the muscle, the loss of elasticity in the lens gradually suppresses the faculty of accommodation.

The excessive innervation required to maintain a state of accommodation at all times for all distances is a potent cause of asthenopia. The hyperopia may exist to such a high degree that the individual cannot exert enough power of accommodation to see clearly at a near distance. Since these patients avoid use of their eyes for near work and seek outdoor employment, they may have no painful symptoms. Ambitious hyperopic persons who use their nerve energy to the point of fatigue or exhaustion are prone to suffer, particularly when the hyperopia is complicated with astigmatism.

For distant vision, the alternative for the exercise of accommodation is to adjust before the eye a convex spherical lens of sufficient power to place the posterior principal focus at the fovea.

Hyperopia is generally classified as manifest, latent and total. *Manifest*

hyperopia is the amount represented by the strongest convex spherical lens which permits clear distant vision. *Total* hyperopia is the amount represented by the strongest convex lens which permits clear distant vision while the eye is at rest or under the influence of a cycloplegic. *Latent* hyperopia is the difference between manifest and total. Total hyperopia can be estimated only by the use of cycloplegia.

The symptoms of hyperopia, including hyperopic astigmatism, are headache, pain in or around the eyes and brow, irritation of the conjunctiva and lids, hordeoli, dim vision, nervousness, and various reflex manifestations. The symptoms arise soon after beginning to use the eyes for close work, and are usually aggravated in proportion as the exercise is exacting or prolonged. These patients usually feel worse by evening, although they may be comfortable early in the day. Some have headache on awaking. The treatment consists of wearing correcting glasses.

Myopia is an optical state in which the retina lies posterior to the posterior principal focus of the dioptric system of the eye (see Fig. 287). The myopic eye is sometimes called a "long eye." Rays originating at the fovea of the myopic eye are projected convergently into space, and intersect at a distance nearer than 6 meters (20 feet).

The point of such intersection and the fovea are conjugate foci. The near-point of the myopic eye is closer than it is in the emmetropic eye, consequently little or no accommodation may be required for near work. Presbyopia is delayed and may never develop. Yet distant vision is never clear without glasses.

Because of "near-sightedness," the myopic individual holds print, sewing and other fine work very near his eyes. This taxes the power of convergence; excessive motor impulses are required to maintain the visual axes in convergence, and this demand on the nervous system is productive of asthenopia. The amount of myopia is represented by the weakest concave spherical lens which permits clear distant vision.

The symptom of myopia is blurred vision for distance, with or without asthenopia. Myopia is associated with astigmatism as a rule, and an examination for glasses should always be made while under cycloplegia. Myopia is treated by appropriate glasses. The condition is often progressive.

Astigmatism is an optical state in which parallel rays of light refracted in the first and second principal meridians do not respectively intersect at the same distance behind the nodal point of the eye. Only one set of them, or neither one, may intersect at the plane of the retina. When one is focused on the fovea, and the other in front of or behind it, the astigmatism is *simple;* when both are focused in front of or behind the fovea, the astigmatism is *com-*

pound; and when one set is focused in front of and the other behind the fovea, the astigmatism is *mixed.*

Simple astigmatism is either hyperopic or myopic, for only one meridian is affected. Compound astigmatism is either hyperopic or myopic, for both principal meridians are affected alike. In mixed astigmatism the eye is hyperopic in one meridian and myopic in the other. While the axes of astigmatism are ordinarily in or near the vertical or horizontal meridian, they are not necessarily so; an axis may occupy any meridian. The two axes of an eye are not always at right angles to each other, although they are rarely otherwise.

Where the axes of astigmatism are obliquely placed, they are generally found to incline symmetrically; that is, both incline either toward the temples or toward the nose. Sometimes the astigmatism of the two eyes, while both may be oblique, will not be at symmetrical axes; one may approach the vertical, and the other the horizontal meridians. One may be hyperopic and the other myopic. A difference in the amounts of astigmatism for the two eyes is not uncommon.

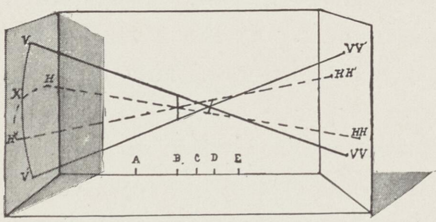


FIG. 289.—STURM'S CONOID.

The principles of astigmatism may be learned from Sturm's conoid. The optics of the figure are easily demonstrated by a contrivance made from an oblong box and four pieces of string. The top and front of the box are removed (Fig. 289). In each end holes are drilled at points marked *V, V', H, H', VV, VV', HH,* and *HH'*. Strings of one color are drawn tightly between the holes *V* and *VV*, and between *V'* and *VV'*. Strings of a different color are similarly drawn between *H* and *HH*, and between *H'* and *HH'*. The strings of one color are represented by solid lines, and of the other by broken lines. The lines represent rays that have undergone refraction.

Of the curved lines at the left end of the box, the solid one represents the vertical corneal meridian, and the broken one the horizontal; their intersection (x) represents the apex of the cornea.

There are five points of interest between the ends of the box; these points depend upon the relative distances between each pair of strings and the strings of each pair. At *A* the broken lines are nearer together than the solid lines; at *B* the broken lines intersect while the solid lines still converge; at *C* the broken lines diverge while the solid lines continue to converge; at *D* the solid lines intersect while the broken lines diverge; and at *E* the solid lines are nearer together than the broken lines.

Figures constructed to touch all four lines at the various points must differ (Fig. 290). At *A*, a vertically oval plane figure fits; at *B*, a vertical line

passes between the solid lines through the intersection of the broken ones; at *C,* a plane circle fits; at *D,* a horizontal line passes between the broken lines through the intersection of the solid ones; and at *E,* a horizontally oval plane figure fits. These figures represent the images as they would be formed on the retina if it were placed at each of the respective points.

At *A,* the posterior principal foci of both sets of lines are behind the image; at *E,* they are in front of it; at *B,* the focus of the broken lines coincides with the position of the image, while that of the solid lines lies beyond it; at *D,* the focus of the solid lines coincides with the position of the image, while that of the broken lines lies anterior to it. At *C,* the image is circular, but since it lies between the foci, it is blurred. The solid lines have not intersected, and their part of the image is upright; the broken lines have intersected, and their part of the image is inverted. At this point the image is mixed.

Since none of the images formed by the conoid are in focus in both meridians at once, all are blurred. The points from *A* to *E* illustrate the essentials of astigmatism, and all cases of regular astigmatism can be explained by them; this is known as Sturm's interval.

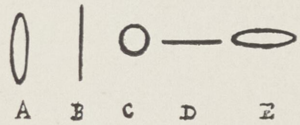


Fig. 290.—Sturm's Figures.

Simple hyperopic astigmatism is represented at *B,* because only one principal focus must be advanced when the retina lies at that point. Should the retina be placed at *A,* the case would be one of compound hyperopic astigmatism, since both foci would have to be advanced. Simple myopic astigmatism is represented with the retina situated at *D,* and compound myopic astigmatism is represented with the retina at *E.* Mixed astigmatism is represented with the retina at *C,* since both foci need adjustment, but in opposite directions to make them coincide in position.

Astigmatism is designated as corneal or lenticular, depending upon the surface of whichever structure is affected. Corneal astigmatism may be partly or wholly compensated by lenticular astigmatism. Accommodation is accomplished by contraction of the ciliary muscle in its whole circumference. In corneal astigmatism that is compensated by automatic lenticular astigmatism, the contraction of the ciliary muscle is segmental; Savage states that the longitudinal fibers (see Fig. 288) accomplish this by tilting the lens. The unnatural impulses required to execute and maintain unequal contraction in the ciliary muscle frequently cause asthenopia or painful sight.

Irregular astigmatism is due to some interruption of continuity in the curvature of a refractive surface, or to a lack of homogeneity in a medium. It often appears in the cornea following pannus, ulcers, abrasions, erosions and injuries. It may appear on or in the lens. Irregular lenticular astigmatism

is due to a lack of uniformity in the refractive power of different segments of the lens.

The symptoms of astigmatism are those of asthenopia or eye-strain. Occipital headaches are commonly found. When the patient attempts to read the letters on the test charts at 6 meters (20 feet), he is prone to confuse *F* and *P*, *E* and *B*, *D* and *N*, *T* and *Y*, *E* and *Z*, *O* and *D*, *C* and *G*, and *S* and *N*. Confusing *F* and *P* is of no importance. In examining the corneal curvature with Placido's disk, the mirrored circles are apt to appear oval. The treatment of astigmatism is by lenses.

METHODS OF EXAMINATION

Notations for lenses are abbreviated. A convex lens is preceded by the plus sign, and a concave one by the minus sign. A sphere is abbreviated Sph. or S.; when the power is indicated, D. precedes the S., thus: + 1.00 D.S. Cylinders are abbreviated by Cyl. or C. Axis is written Ax. or X, followed by the degree at which the axis is placed, thus: + 1.00 Cyl. Ax. 90; the degree sign is usually omitted. The axis marks are indicated on the trial frame (Fig. 291). The sign $\bigcirc$ means "combined with"; it is used to indicate that a cylinder is combined with a sphere or that a prism is combined with a sphere or a cylinder.

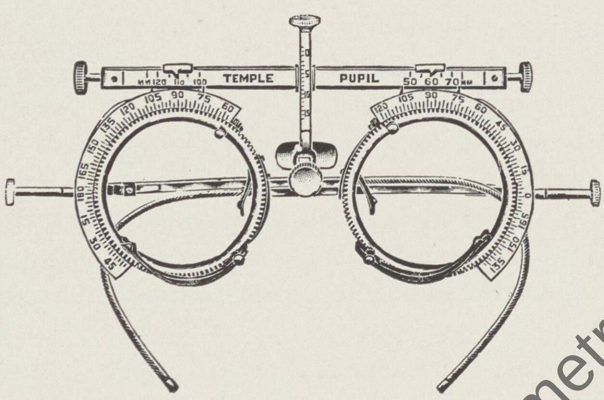


Fig. 291.—Trial Frames.

The Subjective Method.—This test is used in examining applicants for various employments. The patient is seated 6 meters (20 feet) from a well lighted chart of standard test letters (Fig. 292). A trial frame is adjusted before his eyes. An opaque disk is placed in the cell before the left eye, and the examinee names the letters in the line of smallest type that he can read with the right eye. Picture, "broken-ring" and "E" charts (Figs. 293-A and B), graduated in the same way as letter charts, are available for illiterates.

The result for each eye is recorded in the form of a fraction for convenience. The numerator designates the distance between the examinee and the test chart. The denominator denotes the size of the smallest type read. The usual distance is 6 meters (20 feet). The result is recorded $^{6}/_{6}$, $^{6}/_{5}$ or $^{6}/_{10}$ for metric and $^{20}/_{20}$, $^{20}/_{16}$ or $^{20}/_{33}$ for English; these are examples.

The Method by Artificial Myopia.—In hyperopia the ciliary muscle is acting for all distances, and therefore is in a state of contraction to some degree at all waking hours. The muscle possesses power in excess. By putting a strong convex sphere before the eye, more than enough to overcome the hyperopia, that eye does not see clearly while the ciliary muscle is over-acting. As it relaxes, vision improves. The strong convex lens creates a myopic state artificially; the distant vision is dim or foggy, and so the method was called "fogging."

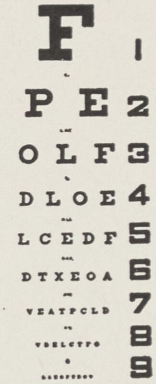


Fig. 292.

The examination is begun as for the subjective test, except that a + 7.00 D.S. is placed before each eye and left for some minutes while the patient looks into the distance. The opaque disk is placed before the left eye. Concave spheres of increasing power are placed before the convex sphere already in the trial frame, as long as they improve vision. The net sphere is the difference between those before the eye, and it is plus or minus according to which is of the higher power. Should the examinee be unable to discriminate between confusion letters, the net spheres are left in the trial frame, and the examination is continued with cylinders.

Various charts have been designed for estimating the amount of astigmatism and the axis at which the cylinder should be placed. The best known of these is the "clock dial"; three straight black parallel lines of equal width, separated by white spaces of the same equal width, extend from near the center of the chart to each of the figures representing the hours on a clock face (Fig. 294).

Fig. 293-A.

The chart is used as follows: The opaque disk is placed before the left eye. The examinee sees one set of lines most clearly, and indicates their direction, as VI to XII, III to IX, IV to X and so on.

A cylindrical lens is placed in the trial frame, before the sphere, with its axis adjusted at a right angle to the lines seen most clearly. If a + 0.50 Cyl. fails to improve the lines seen less clearly, a — 0.50 Cyl. is substituted for it. After finding which one gives the better vision, the power of that cylinder is modified by stronger and weaker cylinders until all lines are seen equally well. The left eye is examined in the same way.

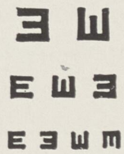


Fig. 293-B.

The letter chart is now substituted for the clock dial; the cylinders are not changed, but the spheres are gradually reduced in the manner already described until the patient reads 6/6 or better. Sometimes a cylinder alone corrects the visual defect.

This method is especially useful in obtaining an approximate correction as a basis for testing the balance of the extra-ocular muscles.

The Dynamic Method is called dynamic because the ciliary muscle is not under the influence of a cycloplegic drug, but has full power to act. The method consists of placing plus and minus spheres, cylinders or spherocylinders of various powers before the eye until the lens which gives best vision is found. This method is not to be confused with dynamic retinoscopy.

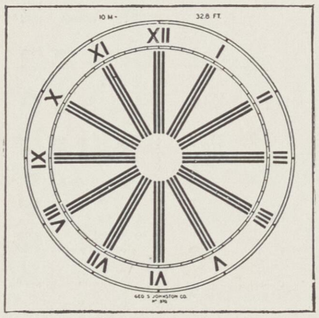


Fig. 294.

The Static Method is called static because the ciliary muscle is at rest under the influence of a cycloplegic drug. The examination is conducted by either the dynamic method or the method of artificial myopia. In prescribing by this plan of examination allowance must be made for the latent power of the ciliary muscle or the effects of cycloplegia.

The Postcycloplegic Method.—Some ten days to two weeks after conducting a test by the static method, after the effects of the cycloplegic drug are exhausted, the patient is examined again. This time he is tested with the lenses which gave best vision by the static method. Ordinarily those lenses will have to be considerably reduced in power.

The Retinoscopic Method.—This is an objective test. It is also called the skiascopic method, and the "shadow test." *Dynamic* retinoscopy applies when no cycloplegic is used, and *static* when the ciliary muscle has been put at rest by a cycloplegic. Retinoscopy may be studied with a schematic eye (see Fig. 121).

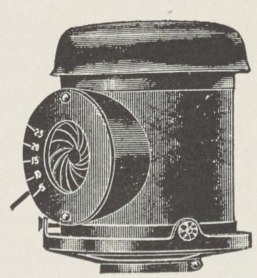


Fig. 295.

Retinoscopy requires an original source of light outside the eye. An electric bulb is placed in an opaque chimney that has a diaphragm shutter on one side even with the bulb (Fig. 295). The shutter is adjustable for a circular opening varying in diameter from 5 to 25 millimeters. This opening transmits light. The best bulbs are those evenly frosted on the inside.

A retinoscope is a circular mirror mounted for use by the hand (Fig. 296). From the center of the mirror the silvering is removed for a space of 1.5 to 3 millimeters in diameter. This unsilvered portion is called the aperture. Mirrors are made plane or concave. The former is generally used. In size the mirror varies from 16 to 40 millimeters (⅔ to 1⅗ inches) in diameter. The smaller is preferable. The mirror is mounted on a nonreflecting black disk.

The patient is seated in a darkened room 1 meter (40 inches) from the examiner. The chimney is placed at the left of the examiner, or behind and above the patient's head. Many prefer to have the chimney near them where they can vary its position at will. The trial case is placed conveniently to the right hand of the examiner. The trial frame is adjusted before the patient's eyes so that the centers of the cells for lenses are situated exactly before the centers of the patient's pupils. After testing the right eye, the frame may have to be readjusted for the left.

The light is switched on. The examiner holds the retinoscope in his right hand with the aperture close before his right pupil, so that he can look through the aperture at the patient, and reflect the light from the chimney into the patient's pupils. Where there are no opacities in the patient's eye or other obstructions to the light, the fundus of that eye is illuminated, and in turn reflects light through the aperture of the retinoscope into the examiner's eye; that fundus becomes a source of light.

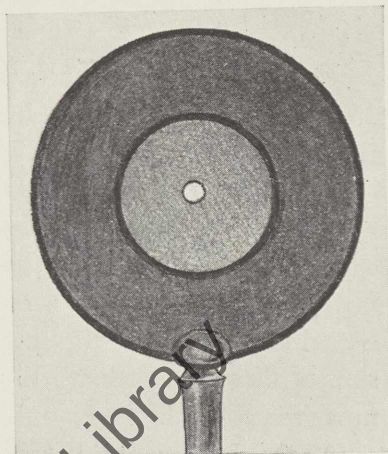


Fig. 296.

The patient must maintain steady fixation in the proper direction. The examiner may place a piece of adhesive tape or white paper on his forehead immediately above the center of his right eyebrow as an object of fixation. The examiner should wear his distance lens correction.

The object of the test is to make conjugate foci of the positions of patient's and examiner's maculæ; the far-point or anterior principal focus of the patient's eye must be made to coincide exactly with the position of the examiner's macula. Thus, at a distance of 1 meter (40 inches) the patient's eye is to be made myopic 1 D. The object is attained by placing before that eye such lens or lenses as will make it 1 D. myopic. The sole exception is the patient who already has a myopia of 1 D.

Since 1 D. of myopia is corrected by a — 1.00 D.S., that amount must be added to the retinoscopic findings to extend the far point to 6 meters (20 feet); this is equivalent to deducting a + 1.00 D.S. An apparent exception sometimes occurs in dynamic retinoscopy, for which no cycloplegic is used. The 1 D. is not deducted previous to the subsequent examination by the test charts; occasionally the findings of retinoscopy are accepted without deduction. This means that the patient was not made myopic 1 D., and that the far-point of his eye was not situated at 1 meter, but at 6; or, that he employed 1 D. of accommodation during the test.

In Figure 297, a ray of light (S) from the chimney strikes the retinoscopic mirror (M) to one side of the aperture and is reflected toward R. When the mirror is rotated about the aperture as a fixed point, S will be reflected toward R'; N and N' are the normals. As the mirror is rotated the location of R is changed; the reflected beam of light can be moved along any meridian of the pupil. When the light is reflected through the patient's pupil, an area of retina is illuminated. This area is indicated by the spaces between the solid black lines at H, E, and M (Fig. 298) on the posterior curvature of the eyeballs. By rotating the mirror, the illuminated area is made to move.

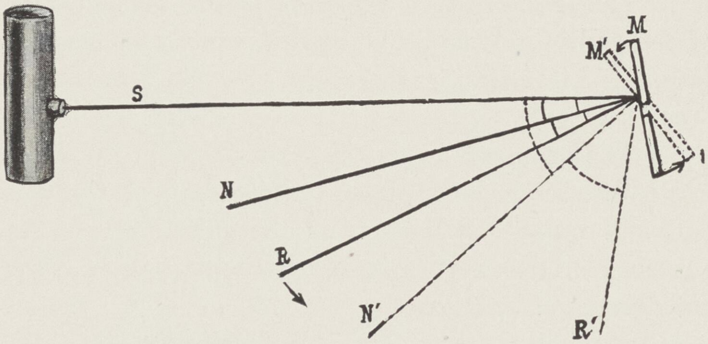


Fig. 297.

In emmetropia (E) and in hyperopia (H), the focal point of the light reflected from the mirror lies behind the eye; the image of the light from the chimney is upright. As the mirror is rotated, the retinal illumination moves in the same direction that the mirror is rotated. In myopia (M), the focal point lies in front of the retina where the black lines intersect, and the image of the light from the chimney is inverted. As the mirror is rotated, the retinal illumination in this case moves in a direction opposite to that in which the mirror is rotated.

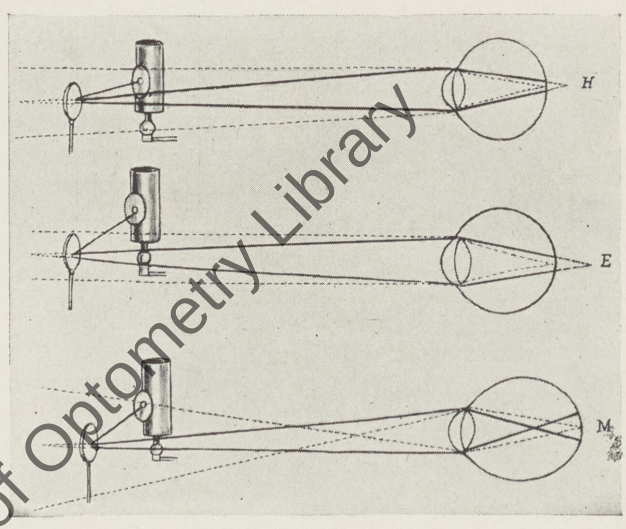


Fig. 298.

Since the retina has become a source of illumination, incident rays arise from it; this is indicated by the diverging dotted lines from a point on the posterior curvature of each eyeball. In emmetropia (E) the lines emerge from the eye parallel, in hyperopia (H) they emerge divergent, and in myopia (M) they emerge convergent. A comprehension of these principles is essential to the relief of ametropia.

Let the light be reflected into the eye *E;* a glow is seen in the pupil. The mirror is rotated so that the light is moved up and down along the vertical meridian of the pupil. The glow in the pupil moves *with* the movement of the mirror. The mirror is then rotated so that the light is moved back and forth along the horizontal meridian of the pupil. The glow in the pupil moves with the movement of the mirror. The same result occurs with a rotation of the mirror in any oblique meridian.

A + 1.00 D.S. is placed in the trial frame in the cell *nearest the eye.* The mirror is rotated as before, but no movement is observed in any meridian. The absence of a movement of the glow in the pupil indicates that the far-point or anterior principal focus of that eye, with the lens before it, is at 1 meter, and that the *point of reversal* has been reached. By adding a — 1.00 D.S., the far-point is removed to 6 meters (20 feet). The spheres neutralize each other and therefore the eye is emmetropic.

Let the light be reflected into the eye *H.* The mirror is rotated in the same manner, and the pupillary glow moves with the movements of the mirror. A + 1.00 D.S. is placed as before; but the light from the pupil continues to move with the mirror. Convex spheres of increasing power are added until no movement is seen in the pupil. The total sphere is then placed in the rear cell of the trial frame. It indicates the strength of the sphere needed to obtain the point of reversal at 1 M. The addition of a — 1.00 D.S. places the far-point at 6 meters (20 feet).

Let the light be reflected into the eye *M.* No movement is seen in the pupil. The far-point of that eye is at 1 meter. The addition of a — 1.00 D.S. places the far-point at 6 meters (20 feet). In this eye, if the movement without a lens is with the mirror, but a convex sphere of less than 1.00 D. brings the point of reversal, then that eye is myopic, but less than 1 D. in amount. If the light moves without a lens, but in a direction opposite to that in which the mirror is rotated, concave spheres of increasing power are added until the point of reversal is reached. The further addition of a — 1.00 D.S. will place the far-point at 6 meters.

Rays from the fovea of *M* are projected convergently; they intersect before arriving at the mirror. This diagram helps to explain why the light in the pupil moves against the rotations of the mirror. The point of intersection must be made to recede with concave spheres until its position coincides with the examiner's macula; then the movement of the light in the pupil will be at the point of reversal.

During the practice of these movements it is necessary that the examiner observe the central area of the pupil. The periphery of the illuminated area may reverse before the central area, or the central before the peripheral area.

This is due to spherical aberration. The tangents of the curved surfaces of a convex spherical lens (Fig. 299) show that as the edge of the lens is approached the tangent or prism angle widens.

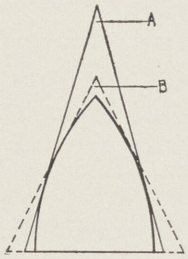


Fig. 299.

Spherical aberration is positive or negative (see Fig. 300, *A* and *B*). In the positive form (*A*) the peripheral rays intersect before the central rays, and so they reverse earlier than the central rays. In the negative form (*B*) the central rays intersect before the peripheral, and so they reverse first.

Spherical aberration is usually present to some degree, and it is confusing to the examiner. The aperture in the chimney should be closed down to 5 millimeters, the position of the light from the examiner's eye and the distance between the examiner and patient must be varied nearer and farther. The central area of the pupil is always kept under observation.

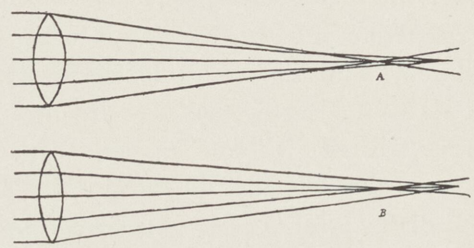


Fig. 300.—*A*, Positive, and *B*, Negative Spherical Aberration.

The Detection and Measurement of Astigmatism.—With a certain sphere, or without a lens, the light in the patient's pupil may be at the point of reversal in one meridian, while in the meridian at a right angle to the first there will be a movement with or against the mirror. This is recognized as a band of light placed vertically, horizontally or obliquely (see Figs. 301, 302, *A* and 303). The detection of the band is easier with the chimney removed some distance from the examiner's eye. When the presence of a band is observed, its axis should be noted by reference to the axes marks on the trial frame.

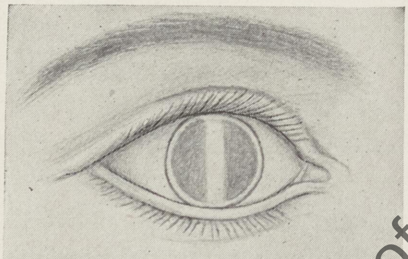


Fig. 301.

When nearing the point of reversal, a faint shadowy ring may be observed surrounding the light in the central area of the pupil. When this ring is circular the presence of astigmatism is unlikely; when it is oval, cylinders are required to make it circular. Occasionally this ring is apparent when a definite band of light cannot be clearly demonstrated.

An erroneous impression of astigmatism is caused by looking through the trial lens at an angle. To avoid this error the examiner must be sure that the patient's line of vision passes through the trial lens perpendicularly to the plane of its surface, or through the optical center of the lens.

A convex cylinder is required to neutralize a band that moves with, and

a concave cylinder to neutralize the band that moves against the mirror. The amount of cylinder needed is determined by trial, as outlined for the sphere. The cylinder must be placed with its axis corresponding exactly to the direction of the band. A cylinder placed a few degrees off axis will apparently change the direction of the band and lead to perplexity.

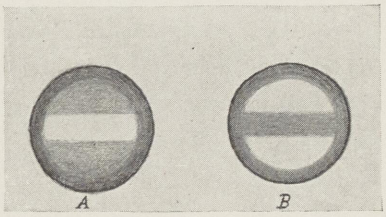


Fig. 302.

A band that moves across the pupil evenly means that the astigmatism is regular. When the band is divided or changes direction, or does not move evenly, the astigmatism is irregular. When moving the mirror vertically, two areas of light, one above and one below, may appear in the pupil (see Fig. 302, *B*); these merge as at *A*. Instead of areas, the light may appear as bands (Fig. 304). These bands are practically always horizontal; the manner of their coming together gave rise to the term "scissors movement."

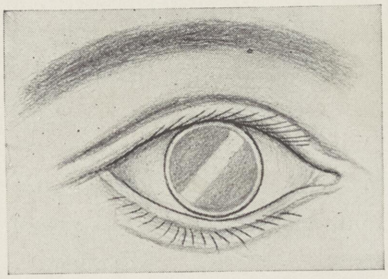


Fig. 303.

The irregular bands which produce the scissors movement may be combined with the band of regular astigmatism. In that event, the scissors bands need not be at a right angle to the meridian of regular astigmatism. After ulcers and injuries of the cornea, the behavior of the light in the pupil may afford little information as to the refraction of that eye. This also is irregular astigmatism.

Retinoscopic findings in irregular astigmatism should be checked with trial lenses and test letter charts; in prescribing for this condition, the trial lenses which give best vision at a postcycloplegic test should be given preference over those found by retinoscopy.

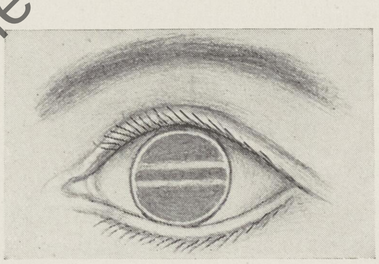


Fig. 304.

Regular astigmatism is usually due to a difference in the curvatures of the principal meridians of the cornea. Irregular astigmatism is usually due to unevenness of the lenticular or corneal surfaces. Corneal astigmatism may be detected and its amount measured by the ophthalmometer, but lenses cannot be confidently prescribed from its findings. It is useful for observing corneal curvature changes in successive examinations.

At the conclusion of the retinoscopic test, the results are recorded for each eye. This is done by a symbol. A vertical line is crossed with a horizontal

line; two of these figures are made, the left one for the right eye, and the right one for the left. The axis marks are placed at the upper end of the vertical or at the left end of the horizontal line. The lens which neutralizes the vertical movement of the light in the pupil is recorded to the right of the vertical and above the horizontal line, and that which neutralizes the horizontal movement is recorded similarly below the horizontal line.

The lenses found after adding a — 1.00 D.S. are not usually the ones that should be prescribed, but they form the basis from which the prescription is calculated.

Some oculists prefer to use the concave mirror in retinoscopy; with it the movements are opposite to those that have been described when using the plane mirror. Electrically lighted retinoscopes are available for those who prefer them.

ROUTINE EXAMINATIONS AND THE PRESCRIBING OF LENSES

The patient has two reasons for desiring an examination; he wishes glasses or a change of lenses on account of defective vision, or he suffers from headache or other symptoms which he or his physician ascribes to eye-strain. A physician sometimes refers a patient for an eye examination in the hope that information may be obtained which will lead to a diagnosis in some obscure illness.

The oculist should conduct his refractions methodically, although a system should be flexible. All data are to be recorded. The examination is begun with a general inspection for proptosis, deformities of the lids, diseases of the lacrimal apparatus, squint, nystagmus, corneal opacities, cataract, iritis, inequalities of the pupils, glaucoma and so on.

The patient is then taken into the dark room, and the vision for each eye is estimated separately by the method of artificial myopia. With the lenses which give the best vision, the muscles are tested for heterophorias and paralyses at 6 meters (20 feet), and also at the reading distance and *position.* Prism divergence and convergence are tested at 6 meters (20 feet).

Without removing the trial lenses, an opaque disk is placed before the left eye, and the near-point of the right is ascertained; the disk is changed to the right eye, and the near-point of the left is determined. When neither near-point is within 33 centimeters (13 inches), convex spheres of increasing power are added to each eye separately, until the near-point of each is brought up to about 28 centimeters (11 inches). The disk is removed and the patient is asked to read with both eyes the smallest type possible on the Snellen hand card. The total lens required by each eye for the reading distance is recorded. These data will be needed to determine the amount of the addition for presbyopia. The trial frames are removed.

The patient is seated facing a lighted window and directed to look as far as possible to the right, left, "up and right," "up and left," "down and right," and "down and left"; this tests the voluntary motor function. The visuomotor function is tested by having the patient fix a moving object as it is carried in each of these directions. The total excursions of each eye are observed and compared.

The convergence power is determined and the cover test is made. The form fields are taken for each eye separately; when these are satisfactory the color fields are not taken. Defective form fields require the taking of color fields, the plotting of Mariotte's blind-spots and other scotomata, and an inquiry into the state of central vision for colors.

The patient is turned from the light and directed to look at some object across the room. The size, shape and equality of the pupils are noted. The direct, indirect or consensual, and convergence reactions of the pupils are noted and compared. The disks are inspected for elevation or marginal cupping.

The corneal sensibility is tested. After phenacain or butyn anesthesia, the intra-ocular tension for each eye is ascertained with the tonometer and *recorded* for all patients who are over thirty years of age. The patient must not raise the tension by compressing the lids against the globe.

Where the form fields are not restricted on the nasal sides, when the corneas are sensitive, where the disks are not cupped at some point of the margin, and when the tonometric readings are within normal limits, cycloplegia may be regarded as safe up to the age of forty years; after forty, mydriasis up to a pupillary diameter of 4 or 5 millimeters is advantageous to the examiner.

Atropin in 0.5 to 2 per cent solutions is used in children up to fourteen or even seventeen years old. The usual preparation consists of instilling a drop into each eye three times a day for one to four days, depending upon the suspected amount of ametropia and its character, whether myopia or hyperopia, and the complications of astigmatism and asthenopia.

Homatropin in 2 per cent solution, to which 2 per cent of cocain may be combined with advantage, is used similarly over a period of twenty-four hours for patients from sixteen to thirty years old. For older patients one drop of the homatropin and cocain solution is instilled in each eye every five minutes for six times, after which the patient waits one hour for the effects. For patients past fifty, one or two drops are sufficient for mydriasis.

Scopolamin hydrobromate in 0.1 to 0.2 per cent solution may be used by instilling one drop in each eye, and repeating it in a half hour. Cycloplegia occurs in about one hour. Scopolamin or hyoscin possesses advantages in the examination of nervous women. When using this drug, pressure should be made over the tear sacs after instilling the drops, to prevent any systemic

reaction. The same precaution should be practiced when using atropin in very blond children, and in those who are susceptible to general reactions from atropin. Colored children usually accept strong solutions without inconvenience.

When satisfactory cycloplegic effects have been obtained, an examination by retinoscopy is made. With the lenses unchanged the patient's attention is directed to a chart of test letters. The spherical lenses are reduced on each eye *separately* after the manner of the artificial myopia method. Cylinders are changed in power or axes only when such changes definitely improve vision. When the retinoscopic examination has been accurately done, the patient should read 6/6 or better with an addition of — 1.00 D.S. to each eye. The muscle balance, not power, is tested again at 6 meters, but not at the reading distance.

The degree of suspension of accommodation by cycloplegia should be tested. The focal point of a + 3.00 D.S. is at 33 centimeters (13 inches), which is the usual reading distance. To the lenses already in the trial frame a + 3.00 D.S. is added before each eye. Where cycloplegia is complete the patient is unable to read small print or standard 50 centimeter block letters nearer than 33 centimeters (13 inches). When young persons and patients who have asthenopic symptoms can read nearer than 30 centimeters, the cycloplegic should be continued for further examination.

Lastly, the eyes are examined ophthalmoscopically. With a plus 16 or plus 12 the crystalline lens is inspected for opacities; with a plus 7, vitreous floaters may be discovered as the patient moves the eye. The fundus is searched with appropriate lenses for evidences of pathological changes in the nerve head, choroid, retina and retinal vessels. Pilocarpin or eserin drops are instilled where homatropin was used in patients who are 40 years old or older.

At the conclusion of the examination the lenses that remain in the trial frame are used as the basis for calculating the strength of the lenses to be prescribed. The cylinders are not disturbed in axes or power. The spheres are subject to change in power. Mainly, they are modified by the state of the muscle balance.

The preliminary examination by artificial myopia affords information as to the strongest lenses that the patient can see through clearly and distinctly at a distance of 6 meters (20 feet). As a general rule, the patient is given the strongest convex or the weakest concave spheres that can be worn comfortably. The full correction is given in cases of considerable esophoria and convergent squint. The weakest correction is given in the presence of exophoria. Spherical lenses cannot be depended upon for the correction of divergent squint.

It is impossible to formulate workable printed rules for the precise use of prisms in the treatment of lateral deviations. These should never be fully

corrected by prisms, since full corrections weaken the muscles. Because of the weight of the lenses, more than a total of four centrads should not be prescribed. Half the total is placed before each eye. They are placed bases out for esophoria, and bases in for exophoria.

Although there are exceptions, it is advisable for the patient to wear the lenses without prisms for two to four weeks. Then the muscle balance is again tested. An improvement in the deviation signifies that relief is possible without prisms. If no improvement has occurred, the patient should be instructed to practice convergence or divergence exercises. After a few weeks another test is made. If desired, the patient may be given atropin to relax convergence or pilocarpin to stimulate it. Atropin is used in 1 : 10,000 to 1 : 20,000 solution, and pilocarpin in 1 : 200 to 1 : 400 solution. The exercises should be continued.

When no improvement has occurred after several months, the physician has the choice of prisms or muscle operation. Each case must be decided on its individual merits.

When a hyperphoria is found at the test given under cycloplegia, it should be corrected, after deducting a half centrad. The required prism is divided between the two eyes when the error is considerable; when the error is small, it is best to place all of the prism base down before the higher looking eye, except in paralytic *hypo*phoria. Hyperphoria is seldom corrected by lenses which contain no prism. Instead of incorporating prisms, lenses may be decentered to obtain a prism effect. The optician will attend to this.

In orthophoria, it is a common practice to deduct half the power of the weaker convex sphere before the one eye, and the same *amount*, not proportion, from the stronger sphere before the other eye. Additions are seldom made to concave spheres, and then only to compel the patient to hold his reading or near work further from the eyes.

The cylindrical correction has not been modified, and the spherical correction to be prescribed has now been determined. In presbyopic patients the near-point for each eye was brought up with additional convex spheres to about 28 centimeters (11 inches). The power of the lenses ascertained by that test, less + 0.25 D.S. for each eye, represents the correction for near work or reading. The difference between the distance and near lenses represents the amount to be *added* as reading segments to each distance lens. Such lenses are called "bifocal." Separate glasses may be prescribed for near use.

It is desirable that the near-points for the two eyes shall be equally distant; therefore the additions need not be identical in amount. For emmetropic eyes, or those made emmetropic by distance lenses, the following additions by attained age usually obtain: At age forty-five add + 1.00 D.S., at fifty add + 1.50 D.S.; at fifty-five add + 2.00 D.S., at sixty add + 2.50 D.S., and

after sixty-five add + 3.00 D.S. In most instances + 0.25 D.S. may be deducted from each of the above amounts. The working distances of tradespeople should be considered in the "addition."

When desired, a postcycloplegic examination may be made before calculating the prescription.

Aphakia means absence of the crystalline lens. The characteristic condition is found after cataract extraction. The normal power of the crystalline lens is represented by + 10.00 D.S. The emmetropic eye, after losing its lens, requires the full + 10.00 D.S. for clear distant vision. The hyperopic eye requires this amount in addition to its native hyperopia. The myopic eye will require no lens when the myopia amounts to — 10.00 D.S., otherwise it will require either a convex or a concave sphere to compensate for the loss of its crystalline.

By reason of the corneal wound made for the extraction of the cataract, these eyes are usually astigmatic, and a cylinder is required. In fitting aphakic eyes with glasses, no cycloplegic is needed. The ametropia is estimated by retinoscopy, and the result is checked at the trial case by the postcycloplegic method.

Directions for making frame measurements are deliberately omitted. When one wishes to order complete glasses, he should obtain a few sets of fitting frames and obtain the measurements from them. However, it will be necessary to measure the pupillary width in each case. This is the distance in millimeters between either the right borders or the left borders of the pupils (see Fig. 244).

Some patients accept a cylinder with a horizontal axis before one eye, and no cylinder, or a cylinder of opposite sign at the same axis, or a cylinder having the same sign but with axis vertical before the other eye. No trouble is experienced when looking through the optical centers of the lenses. When looking through the lower halves of the lenses, in the reading position, the cylinder with a horizontal axis becomes a prism. This produces hyperphoria, which will be attended by the symptoms of vertical imbalance of the muscles. A compensating prism may be ground into or cemented onto the lower half of either lens.

These patients should have their hyperphoria tests in near vision made in the reading *position,* and not on a level with the eyes.

Myopia is sometimes progressive up to early adult life. These patients should be relieved of the strain of accommodation, and it is advisable to undercorrect them by 1 diopter or 2 diopters, rather than prescribe separate glasses for distant and near use. A child may not be depended on to make the change.

CHAPTER XIX

INDUSTRIAL INJURIES, THEIR COMPENSATION AND SIMULATION

The inauguration of an era for the payment of compensation for industrial injuries and occupational diseases led to the adoption of a variety of procedures by the several states for the adjustment of losses due to impaired visual efficiency. The Section on Ophthalmology of the American Medical Association in 1919 appointed a committee to investigate and study the subject, and to recommend routine methods by means of which uniformity in awards might be established. The 1925 report of this committee was adopted by the Section and approved by the House of Delegates. The committee made a supplementary report in 1927. These reports are used authoritatively in the following discussion.

The first necessity was to outline a method by which to determine the percentage of visual efficiency *retained* by an individual whose visual functions were impaired in consequence of occupational disease or injury. The difference between the retained per cent and 100 would represent the percentage of visual loss. The recommended method facilitates the computation of impairment, whether one or both eyes are affected. Visual efficiency, industrially considered, is based on three coördinating functions or factors which are measurable, and on which percentages may be calculated. These are the acuity of central or macular vision, the field of vision, and the muscle or motor function for maintaining binocular single vision in the field of binocular fixation. Other defects may be present, but they are not measurable.

The second necessity was to determine maximum or 100 per cent, and minimum or 0.0 per cent limits for each of the three factors. Distant vision of $6/6$ (meters) or $20/20$ (feet), and near vision of $35/35$ (centimeters) or $14/14$ (inches) represent the maximum. The minimum in distant vision is placed at $6/240$ (meters) or $20/800$ (feet), and $35/1400$ (centimeters) or $14/560$ (inches) represents the minimum in near vision.

The field of vision (see Fig. 123) is considered maximum when its boundaries extend from the point of fixation temporally 65 degrees, temporally and down 65 degrees, down 55 degrees, nasally and down 45 degrees, nasally 45 degrees, nasally and up 45 degrees, up 45 degrees, and temporally and up

55 degrees. The sum of these figures is 420, and this number represents 100 per cent efficiency for this factor. The minimum limit exists when there is a concentric contraction to 5 degrees.

The maximum motor efficiency is represented by the absence, and the minimum by the presence of binocular diplopia in all parts of the field of binocular fixation. This is the only part of the examination in which one eye or the other is not covered.

The third necessity was to devise methods of examination whereby intermediate percentages could be determined. Central visual acuity for distance is tested by standard test charts * (see Figs. 292, and 293-A and B) at 6 meters (20 feet). The characters on these charts are standard blocks (see Fig. 284) of 5 minutes in each dimension for the individually designated distances. Central visual acuity for near is tested by standard Snellen and Jaeger cards at 35 centimeters (14 inches).

Table A does not differ essentially from the one submitted by the committee in 1927, but it is modified in arrangement for the purposes of this text. The first and second columns contain the notations for distance vision at 6 meters and at 20 feet; the third and fourth columns contain the notations for near vision at 35 centimeters and at 14 inches; the fifth column contains the Jaeger type equivalents for the Snellen near vision notation; the sixth column states the visual angles for the different sized characters at the fixed distances at which the tests are made; the seventh and eighth columns respectively specify the central visual efficiency and impairment.

The efficiency of central visual acuity for near is reckoned at twice the value of that factor for distance. In computing the efficiency of one eye the distance acuity is multiplied by 1, and the near by 2; the sum of the products is divided by 3. If the distance acuity is 60 per cent, and the near is 36 per cent, then:

$$\frac{(60 \times 1) + (36 \times 2)}{3} = \frac{60 + 72}{3} = \frac{132}{3} = 44 \text{ per cent.}$$

The impairment of this factor in this case amounts to 56 per cent.

The test shall be made with correcting lenses for each eye, if vision can be improved by their use. However, the injured eye, or the less efficient eye if both are injured, shall not be tested with a lens that differs by more than 4 diopters in spherical power from that which gives best vision to the more efficient eye. When the latter eye sees best with a + 2.00 diopter sphere, the less efficient eye may see best with a — 3.00 D. S., but it shall be tested with a — 2.00 D.S. because that lens differs by not more than 4 diopters

* A chart endorsed by the committee is now obtainable from wholesale opticians.

Table A.—Central Visual Acuity Efficiency.

Snellen Notation for Distant Vision		Snellen Notation for Near Vision		Jaeger Type	Visual Angle	Percentage	
						Efficiency	Loss
6/6	(20/20)	35/35	(14/14)	No. 1	5.0	100.0	0.0
6/9	(20/30)	35/52.5	(14/21)	No. 2	7.5	91.4	8.6
6/10	(20/33)	35/60	(14/24)	No. 3	8.5	89.5	10.5
6/12	(20/40)	35/70	(14/28)	No. 4	10.0	83.6	16.4
6/15	(20/50)	35/87.5	(14/35)	No. 6	12.5	76.5	23.5
6/18	(20/60)	35/105	(14/42)	No. 8	15.0	70.0	30.0
6/21	(20/70)	35/122.5	(14/49)	No. 9	17.5	63.9	36.1
6/24	(20/80)	35/140	(14/56)	No. 10	20.0	58.5	41.5
6/30	(20/100)	35/175	(14/70)	No. 11	25.0	48.9	51.1
6/36	(20/120)	35/210	(14/84)	No. 12	30.0	40.9	59.1
6/42	(20/140)	35/245	(14/98)	No. 14	35.0	34.2	65.8
6/48	(20/160)	35/280	(14/112)	No. 16	40.0	28.6	71.4
6/60	(20/200)	35/350	(14/140)	No. 17	50.0	20.0	80.0
6/72	(20/240)	35/420	(14/168)	No. 18	60.0	13.0	87.0
6/96	(20/320)	35/560	(14/224)	No. 19	80.0	7.2	92.8
6/144	(20/480)	35/840	(14/336)	No. 20	120.0	2.0	98.0
6/180	(20/600)	35/1050	(14/420)	.....	150.0	0.6	99.4
6/240	(20/800)	35/1400	(14/560)	.....	200.0	0.1	99.9

from a + 2.00 D. S. Each eye must be corrected and tested while the other is covered.

The test charts shall be illuminated by not less than 3 nor more than 10 foot-candles. A foot-candle expresses the amount of direct illumination obtained at a distance of 1 foot from a sperm candle burning at the rate of 8 grams (120 grains) per hour.

The visual field shall be taken on a perimeter along the eight radii already indicated. The test object shall be white, 1 degree in diameter, and illuminated by not less than 3 foot-candles. For the Schweigger perimeter (see Fig. 126) the test object will be 2.5 millimeters (1/10 inch) in diameter. The sum of the eight radial measurements is to be divided by 420, and the quotient will represent the efficiency of this factor in percentage. Should the sum of the radii amount to 252, then: 420 : 252 : : 100 : *x*, or 60 per cent. The impairment of the visual field in this case is 40 per cent.

The motor function shall be examined by means of a chart 20 degrees square; it is laid off into twenty rectangles, each of which is 4 degrees high and 5 degrees wide. At a distance of 1 meter (40 inches) the chart will be 35 centimeters (14 inches) square. The point of fixation is located 2 degrees above the center of the chart (Fig. 305). The examinee is seated so that he can fix on the point of fixation while his eyes are in the *primary position* (see

Fig. 230); his head must not be moved during the test. The trial frame is adjusted and a red glass is placed before one eye. The examinee fixes on a small light as it is carried successively into each of the twenty rectangles. The number of these in which diplopia occurs is recorded as the numerator of a fraction whose denominator is 20.

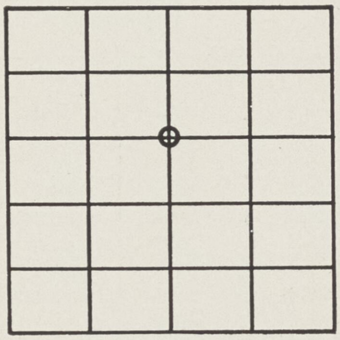


Fig. 305.

The efficiency rating of this factor is based on the fraction, as in Table B. The first column denotes the proportionate number of rectangles in which diplopia was found. The second column specifies the percentages of efficiency; the differences between these percentages and 100 equals the percentages of motor impairment.

Should diplopia be found in seven rectangles and no more, the efficiency would be 81 per cent, and the impairment 19 per cent.

Table B.—Motor Field Efficiency.

0/20	100	11/20	67
1/20	98	12/20	63
2/20	95	13/20	59
3/20	92	14/20	55
4/20	89	15/20	50
5/20	87	16/20	45
6/20	84	17/20	39
7/20	81	18/20	32
8/20	77	19/20	22
9/20	74	20/20	0
10/20	71		

Industrial visual efficiency is represented by the product of the *efficiency* percentages of the factors of central visual acuity, visual fields, and the motor field. In the illustrations given, the central acuity was 44 per cent, the visual field was 60 per cent, and the motor field was 81 per cent; then: $0.44 \times 0.60 \times 0.81 = 0.21384$, or 21.384 per cent visual efficiency, and 78.616 per cent impairment.

When both eyes are injured, the industrial visual efficiency of the *individual* is computed by multiplying the percentage efficiency of the less efficient eye by 1, and that of the more efficient eye by 3. The sum of the two products is divided by 4, and the quotient will represent the industrial visual efficiency of the individual. If the efficiency of one eye is 20 per cent, and that of the other is 40 per cent, then:

$$\frac{(20 \times 1) + (40 \times 3)}{4} = \frac{140}{4} = 35 \text{ per cent};$$

the visual impairment in that case will amount to 65 per cent.

The visual efficiency of an individual who has an impairment of one eye only is computed similarly:

$$\frac{(20 \times 1) + (100 \times 3)}{4} = \frac{320}{4} = 80 \text{ per cent efficient.}$$

While various effects of an injury may exist, only the three factors already discussed can at present be calculated with mathematical precision. These other defects, such as are due to deformities of the lids, impaired accommodation and so forth, are properly considered in the award, but the award must not exceed the sum which is allowed by law for total permanent disability.

Visual impairment may have existed prior to injury, but evidence of such previous impairment shall not be taken into account in calculating efficiency. It is properly recognized in the award. A visual acuity known to be better than $6/6$ before injury, but which is reduced to $6/6$ by injury, is not considered as having been impaired, since the reduction does not depress the acuity below the maximum limit. This observation is consistent with the recommended maximum limits for the visual field, since these limits are below the average normal limits.

Permanent disability should not be conceded until the resources of medical science have been satisfied.

The tests for visual impairment are subjective. The cupidity of the examinee may influence the results. The examiner should be familiar with the opportunities for simulation and exaggeration.

TESTS FOR MALINGERING AND SIMULATION

An individual may assume disability for any reason that promises an advantage. The detection of feigned blindness is often difficult because the tests are largely subjective, and because an individual can be trained to give normal responses. Generally it is alleged that only one eye is blind or defective, and frequently the degree of disability is exaggerated. It is advisable to allow the examinee to understand that his statements are accepted, but that loss of vision must be established by proof.

By reference to Figures 122 and 256 it can be seen that a lesion in the visual pathways posterior to the lateral geniculate body can cause blindness without effect on pupillary reactions. As a rule, the reactions of the pupil of a blind eye do not equal those of a seeing eye, and they may be absent. They

should always be observed and compared. An eye may be blind without visible abnormality in the fundus, and no person should be accounted a malingerer because the fundus appears to be healthy. Old injuries must be looked for, and their possible relation to an existing disability must be estimated. A hole in the iris with a corresponding corneal opacity is an example.

The lenses that represent the distance and near corrections are to be used in the trial frame for the several tests. While conducting these, the examiner must constantly watch both eyes and be ready to circumvent any attempt to close the alleged bad eye in order to see how things look to the good one.

1. The examinee is seated for the distance test. To the glasses in the trial frame a + 0.75 cylinder and a — 0.75 cylinder are placed before the good eye with their axes coinciding at either 45 or 135 degrees. While the examinee is reading the chart, one cylinder is slowly rotated. A difference of 30 degrees in the axes of the cylinders is sufficient to distort the smaller letters. If the reading is not interrupted, the bad eye can see. The cylinders will need to be stronger than 0.75 when their axes are placed at either 90 or 180 degrees.

2. To the distance lenses a + 5 diopter sphere is added before the good eye and a + 0.13 diopter sphere before the bad eye. Reading the small letters proves that the bad eye sees.

3. While the examinee looks at a distant light a 6 centrad prism is placed base out before the bad eye; if it has sight the cornea will rotate in to avoid diplopia, and it will also rotate out when the prism is removed. This is an objective test. When viewed through the prism the cornea will always seem to rotate; it should be observed from the side and behind the prism.

4. Neither eye is blind when bar reading can be done rapidly.

5. To the distance correction a + 5 diopter sphere is placed before the good eye and a + 3 diopter before the bad eye. Fine print is held at 20 centimeters (8 inches), the focal distance of the + 5 lens, and the examinee is directed to read rapidly. The print is slowly withdrawn; continued reading much beyond 20 centimeters means that the bad eye can see.

6. The examinee is given print and directed to read rapidly; a 2 centrad prism is suddenly placed base down before the bad eye. Hesitation and confusion suggest that a vertical diplopia has been created, and diplopia means that both eyes can see.

7. Arabic numerals and letters are arranged on each face of a white card like vertical columns of figures; parts of the characters are in black and parts are in red. A red glass is placed before the good eye and a green one before the bad eye. The examinee is directed to read the characters rapidly as soon as they are exposed. The two faces of the card can be presented alternately. When the red and black characters are read equally well, both eyes have vision.

8. A serious operation is proposed. A malingerer usually rejects the offer promptly.

The simulation of bilateral blindness is unusual. The pupillary reactions should be tested. The examinee is directed to perform voluntary rotations; a truly blind person usually does this well. A malingerer will attempt to move his eyes aimlessly, and his mind is occupied with that idea. While thus engaged he is suddenly told to look straight ahead. The light from a hand ophthalmoscope is switched on and a 6 centrad prism is placed base out before one eye and then removed; the test is made on the other eye similarly. If either eye rotates to the prism it does so to avoid diplopia.

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